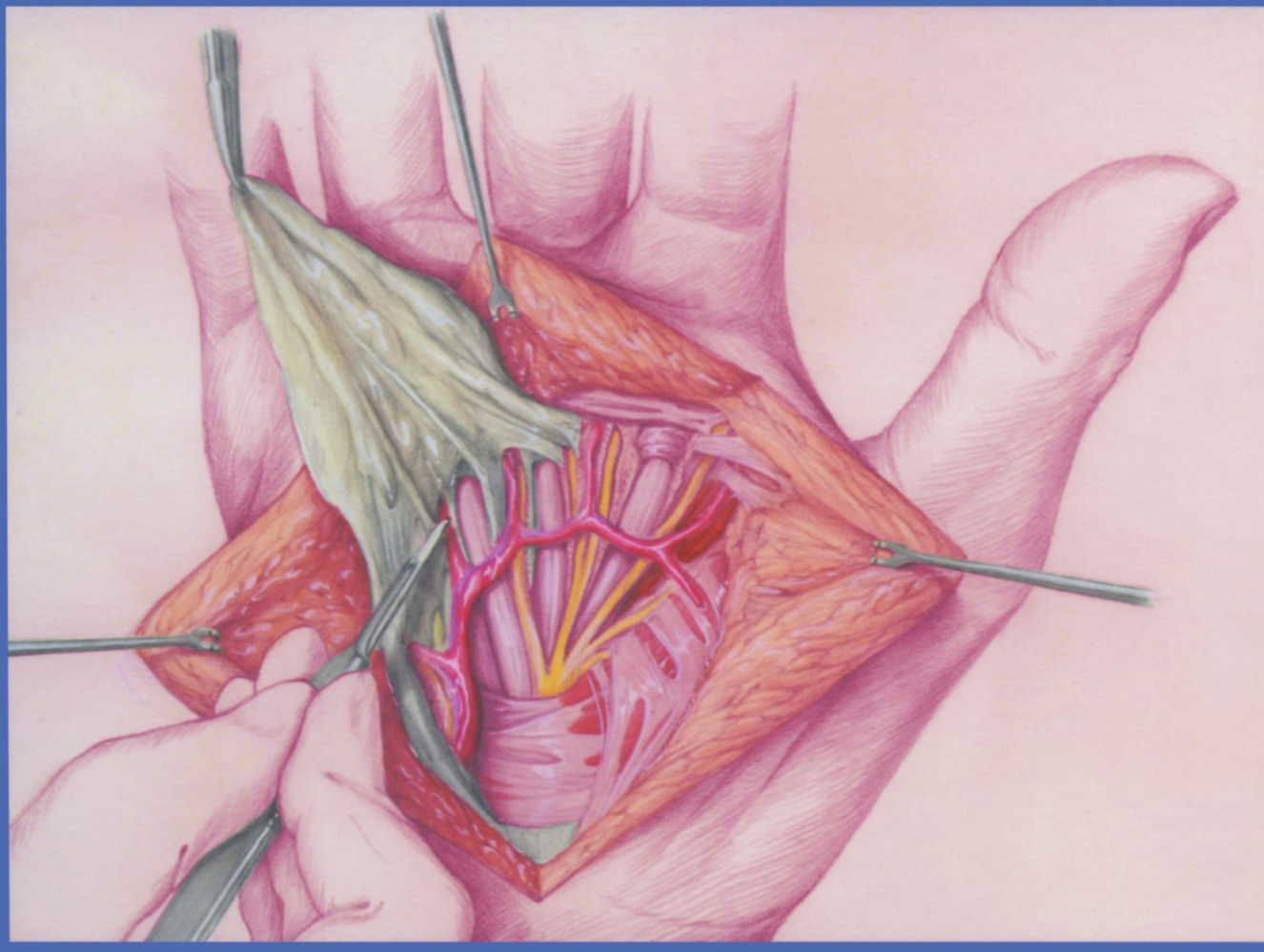
peter brenner, ghazi m. rayan

dupuytren's disease

a concept of surgical treatment

foreword by hanno millesi



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Dupuytren's Disease
A Concept of Surgical Treatment

Foreword by Hanno Millesi
With Illustrations by Jakob Gratzer
and Hanna Schimek

Springer-Verlag Wien GmbH

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Foreword

Dupuytren's disease is a fascinating disorder. It does not match any known textbook pattern: It is neither inflammatory in nature, nor a tumor. In addition, it is not induced by trauma, and it does not represent a malformation in the strict sense of the word, although hereditary factors do play a role.

Every year, more than 30 papers are published on this topic. Any etiological or pathological factor imaginable has been made responsible. When the field of bacteriology was in its heyday, a specific pathogen was believed to cause Dupuytren's contracture. Later, the blame was obviously on a virus; and today I feel reassured to learn from this excellent book that there is a relationship between HIV and Dupuytren's disease.

During the thirties and forties, nearly all surgeons of later fame published some paper on Dupuytren's disease. A small number of these surgeons stuck to this topic and dedicated most of their life's work to the study of Dupuytren's disease, as Dr. Brenner and myself did.

Anyone who has been engaged in the study of Dupuytren's contracture for any length of time develops "unbounded enthusiasm", delves deeply into the subject matter, and finally publishes something like a "bible". Just like this book is the "Bible of the Glycosaminoglycan Theory".

I am convinced the reader will not only be thrilled with this theory, but also become quite excited about Dupuytren's disease. Please remember, however, that there are many different convictions. Don't take a single one of them as the be-all and end-all.

Vienna, May, 2001

Prof. Hanno Millesi, MD PhD

Preface

Digitopalmar flexion contracture is never just an affliction of the extremities; in reality, Dupuytren's disease represents a systemic connective tissue malady. This is confirmed not only by subtle biochemical changes that are not immediately visible, but also by obvious ectopic fibrous deposits. These accumulations of connective tissue can be located above the dorsal proximal interphalangeal joints, on the auricular helix, the elbow, the sole, and inside the penis, and they are histologically identical. Efforts to find a single cause of this generalized disorder have failed.

Countless hypotheses concerning the etiology of Dupuytren's disease have been published. We focus only on the most controversially debated views, which are important for the choice of a surgical procedure on the one hand and a potential systemic treatment on the other hand.

Thanks to my coauthor, G. A. Rayan, head of the Department for Hand Surgery at the University of Oklahoma/USA, the chapter entitled, "Anatomy of the palmar fascia" will clarify that modern examination techniques, such as arthroscopy and microdissections using an operating microscope, lend credibility to the term "connective tissue corpus" introduced by GRAPOW. It is now justified to refer to the communicating microarchitecture of the underlying connective tissue skeleton between the palm and the fingers, or the digitopalmar continuum, or the fascial complex, respectively.

This chapter also illustrates discrepancies between the clinical and the anatomical nomenclature. In the anatomical nomenclature, for instance, there are no terms equivalent to "lateral digital sheet" or "vertical fibers of the skin anchoring system". The term "fascia" in its rigorous anatomical sense is strictly reserved for "layers, which cover muscle." In the Angloamerican literature, clinicians use this term also to describe the digitopalmar continuum.

If all these recent anatomical findings are put into practical use, one will clearly achieve better outcomes with respect to hand function, which can be assessed with the "coefficient of improvement" according to TUBIANA. This is clearly supported by our own clinical results and microsurgical dissections.

The variety of treatment methods are not competing with each other, because each of them has a set of well-founded indications. Needle "fasciectomy" to treat patients with rheumatic diseases who are on steroids has its indications, as much as fasciotomies are reserved for the elderly with hygiene problems of the hollow of the palm and stage IV. In radial types or expansive ulnar types of Dupuytren's disease, total fasciectomy will be inevitable, but currently, partial fasciectomies are used more often. Because of its low rate of complications, the "open palm technique" holds a prominent position.

MOERMANS' minimal invasive segmental aponeurotomy was popularized in Belgium and seems to be prevailing. The other end of the treatment spectrum includes radical methods, such as dermatofasciectomy, sometimes already used as the primary operation, in cases of familial diathesis and intradermal infiltration.

Apart from the original model developed by MESSINA, other methods of continuous elongation techniques (T.E.C.), including the less traumatic balloon technique by PIZA-KATZER, will be presented. In addition to individual surgical techniques, common complications, rates of progression or relapse will be presented, to assess the aforementioned methods.

Based on the plasma distribution pattern of sulfated and non-sulfated glycosaminoglycans, Dupuytren's disease can be diagnosed with a probability of 91.7% by laboratory methods alone, and without knowledge of the patient and his or her connective tissue disorder. We are hoping that in the future,

glycosaminoglycan determination will become as commonplace with respect to diagnosing Dupuytren's disease, as the rheumatoid factor test is today with respect to other connective tissue diseases.

In addition to excessive concentrations of hyaluronic acid, which is indicative for repetitive remodelling, the fascial extracellular matrix shows changes in the relation of collagen I/collagen III and of the embedded mobile and stationary cells. The chameleon-like myofibroblast seems to be *the* pathognomic cell in Dupuytren's disease. Myofibroblasts are very sensitive to oxidative stress. Their synthesis and cell surface behaviour change and modify the extracellular matrix in which the myofibroblast itself exists. This is an exceptional autocrine or paracrine situation that is being perpetuated by fibroblast growth factor and transforming growth factor subunit beta.

It is therefore not surprising that Dupuytren's disease is almost unknown in regions where large amounts of antioxizing citrus fruit, native olive oil, and a high proportion of raw food are consumed – for example, in the Mediterranean or Asia.

We will show to which extent scavengers, inhibitors of cell-matrix interaction, and even somatic gene therapy may offer useful treatments in the future. Those working actively in surgery will thus encounter a basic excursion into cellular biology and biochemistry, as far as both relate to Dupuytren's disease.

Jakob Gratzer has translated my ideas into telling illustrations. His artistic contribution is a valuable and characteristic feature of this book.

This book is meant for all those interested in hand surgery, may they be hand surgeons, plastic surgeons, orthopaedic surgeons, traumatoligists, general surgeons, rheumatologists, or be involved in basic research. Please feel free to contact us, if you would like to make any suggestions of additions.

Leuven, September 2002 Peter Brenner

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Abbreviations

ADM	Abductor digiti minimi
ADP	Adenosine diphosphate
AER	Apical ectodermal ridge
AGE	Advanced glycation end products
AIDS	Acquired immune deficiency syndrome
AUDIT	Alcohol use disorders identification test
bFGF	Basic fibroblast growth factor
CD 68	Indicator of human, mature tissue macrophages
CDC	Centers for Disease Control
CI	Coefficient of improvement
Ch-0-S	Chondroitin
Ch-4-S	Chondroitin-4-sulfate
Ch-6-S	Chondroitin-6-sulfate
D-0-S	Dermatan
D-4-S	Dermatan-4-sulfate
D-6-S	Dermatan-6-sulfate
DIP	Distal interphalangeal joint
DNA	Desoxyribonucleic acid
EGF	Epidermal growth factor
EFGR	Epidermal growth factor receptor
FGF	Fibroblast growth factor
FLS	Fibrous long-spacing collagen
25 F 9	Indicator of late inflammatory phase
GAG	Glycosaminoglycan
Gamma-GT	Gamma-Glutamyl Transferase (GGT)
HA	Hyaluronic acid or hyaluronan
HLA	Histocompatibility antigen
IFN	Interferon
IPD	Intermittent pneumatic distraction
IPPL	Interpalmar plate ligament
IVRA	Intravenous regional anesthesia
LCA	Leucocyte common antigen
LDS	Lateral digital sheet
LETS	Large, external transformation-sensitive protein
LJM	Limited joint mobility
LPA	Lysophophatic acid
MP	Metacarpophalangeal joint
n	Amount (number)
NL	Natatory ligament
PA	Palmar aponeurosis
POD	Postoperative day
PDGF	Platelet derived growth factor
PDGFR	Platelet derived growth factor receptor
PFC	Palmar fascial complex
PIP	Proximal interphalangeal joint
RM 3/1	Indicator of intermediary phase of inflammation (recovery phase)
ROM	Range of motion
RSD	Reflex sympathetic dystrophy
s	Standard deviation
SNAP	Sensory nerve action potentials
T.E.C.	Tecnica di Estensione Continua
TFG	Transforming growth factor
TFG β	Transforming growth factor subunit β

1 Introduction

The digitopalmar contracture named after DUPUYTREN (1831, 1832), is a fibrous, proliferative hyperplasia of pre-existing connective tissue structures of the fascia of the fingers and palm, also called the digitopalmar continuum (Fig. 1a and b) (BRENNER, 1997).

As many as 1.9 million people in Germany suffer from Dupuytren's disease (BRENNER et al., 1994; BRENNER et al., 2001).

This sheds light on the economic consequences as well as on the individual suffering because of the restrictions in the use of the afflicted hands. Not only does the disorder affect the patients' work-life—insecure grip and increased risk of accidents come to mind—but also their private life, especially in cold northern climates—particularly if the flexion contracture prevents them from putting the hand into a trouser pocket or from donning gloves during the winter (BRUNER, 1987).

Beyond that, Dupuytren's diathesis leads to grotesque flexion contractures of both hands, and often to distant ectopic spread with multiple connective tissue deposits. This can be observed even in young adults.

It is this book's aim to compile the currently known aspects of digitopalmar flexion contracture and to reason that Dupuytren's disease is indeed a systemic connective tissue disease.

The current surgical treatment options will be presented with their specific complications, as well as the associated recurrence and progression rates, permitting a therapeutic valid comparison of various techniques.

Suggestions on how to treat secondary contractures will conclude this compendium.

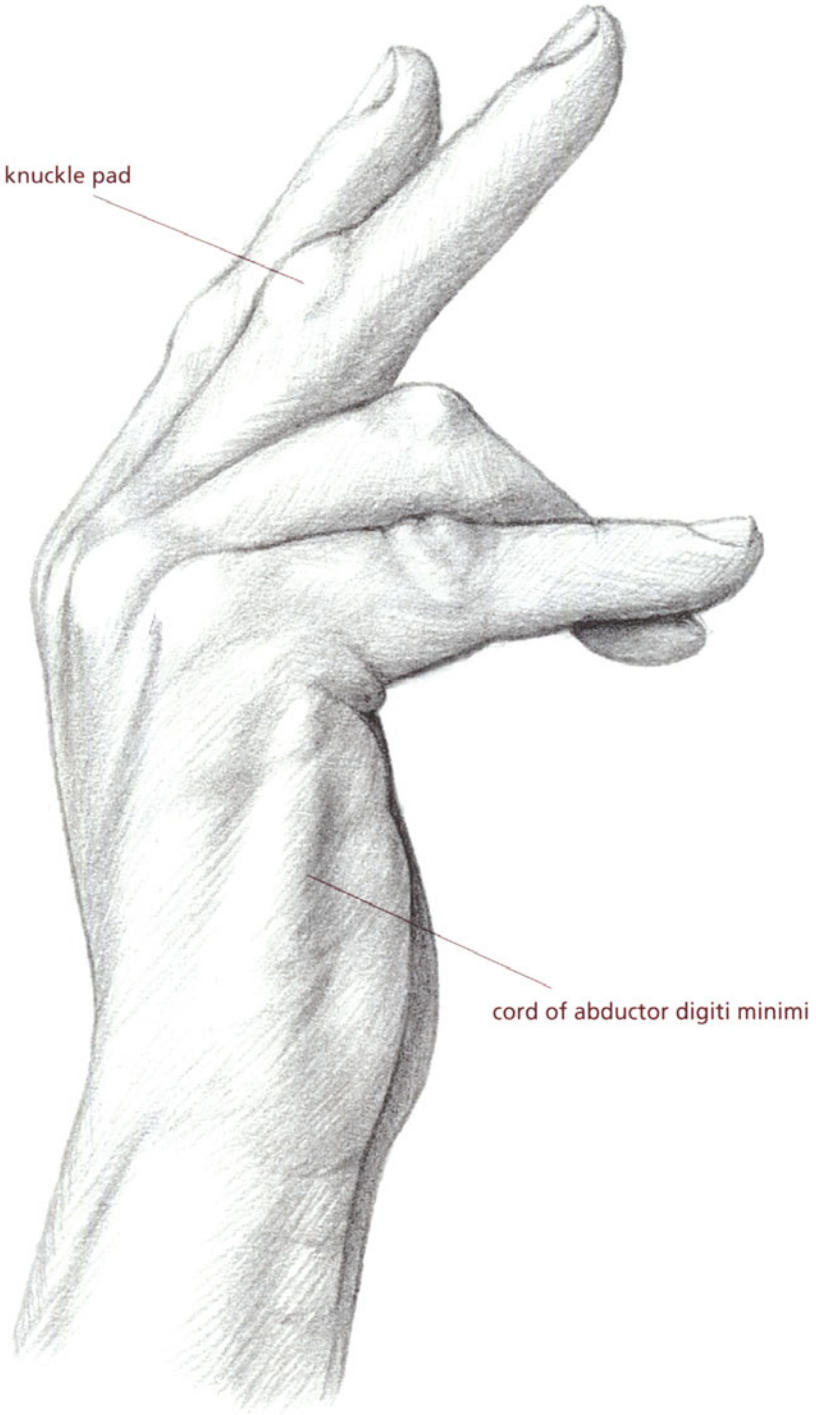


Fig. 1a. Established digitopalmar flexion contracture of the ring and little finger, and starting on the middle finger (ulnar type of Dupuytren's disease), side view.

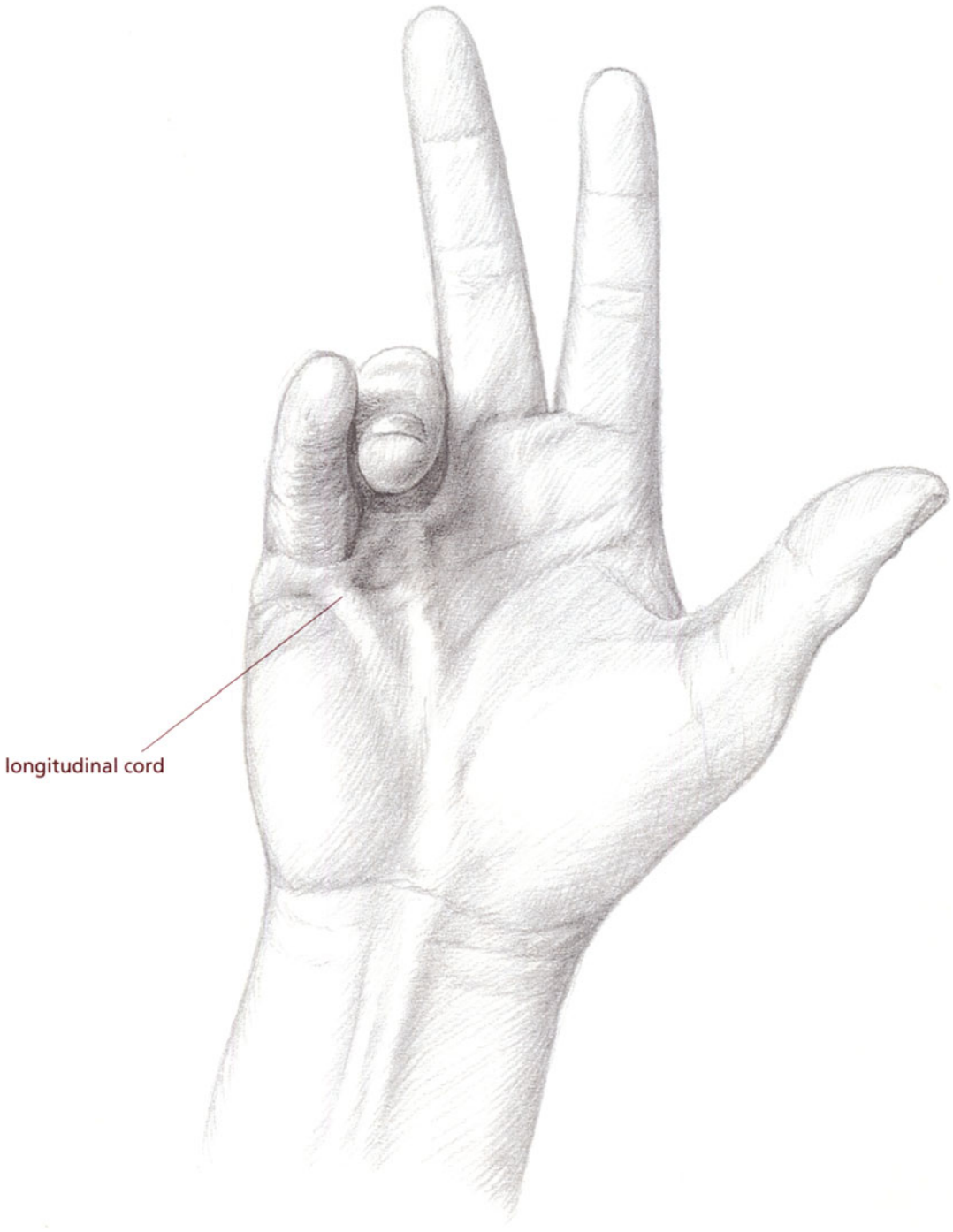


Fig. 1b. View of the right hand of the same patient. Cord stage of Dupuytren's disease with puckered palmar skin. The ring finger extension deficit is 140 degrees and that of the little finger approximately 85 degrees. The middle finger cannot be extended fully.

2 Dupuytren's disease—a historical overview

The clinical details of digitopalmar contracture—now known as Dupuytren's disease, and topic of the famous lecture at Hôpital-Dieu in Paris on 5 December 1831 by the French anatomist and surgeon Guillaume DUPUYTREN (1777–1835; Fig. 2), had been described in much earlier publications (COOPER, 1824; PAILLARD and MARX, 1831; DUPUYTREN, 1831, 1832, 1834).

Fig. 2. The anatomist and surgeon Guillaume DUPUYTREN (1777–1835) in the atrium of his workplace, Hôtel-Dieu, in Paris.

For the first time, the disorder was mentioned in the 12th century in the Longer Saga of Magnus of Orkney (Scotland), where a crippling contracture of the fingers is described. A pilgrimage to the shrine of Holy Magnus had allegedly resulted in complete recovery and unrestricted extension of the affected fingers. The manuscript reads that the fingers became "supple" and "flexible" and could be put "to any use" (WAHLEY et al., 1993, 1994).

Later the cosmopolitan and designated city doctor of Basle, Felix PLATTER (1614), mentions the disease, and this is the first time it is mentioned in the history of medicine (DAHMEN et al., 1967; GLAS et al., 1977). The most striking feature, according to the author, were the "digiti astrici." He wrongly thought that "contractio digitorum" is caused by a displaced flexor tendon.

In 1777—coincidentally Guillaume Dupuytren's birth year—Henry CLINE dissected two hands of a corpse, which had flexion contractures. He documented the striking finding in his notebook, which still exists today, albeit in fragmentary form (ELLIOT, 1988).

In agreement, Napoleon's physician, Alexis BOYER (1818) called the disorder "crispertura tendinum," according to the prevailing view of those times that the cause was a condition affecting flexor tendons and sheaths (GRAPOW, 1887; GOSSET, 1985; ELLIOT, 1988).

The Englishman Sir Astley COOPER (1824) ascribed the disorder primarily to the palmar fascia. It is extraordinary and remarkable that COOPER (1824) published his findings about a decade before DUPUYTREN. The condition is, however, eponymically associated with the latter (DUPUYTREN, 1832; PELTIER, 1958; LETTIN, 1964; ACKERKNECHT, 1967; NIGST, 1971; BOUCHET, 1986; LURIE et al., 1987; SALZBERG et al., 1987; NIGST, 1991).

Initially, the flexion contracture was graphically named "apostle's hand" or "oath hand" (JAMES et

al., 1952; HUESTON, 1960). WEBER (1978) pointed out the cultural history of the extension deficit of the ulnar side from Dupuytren's disease in fine arts and ritual, mostly Christian, gestures and actions, for example, in the papal sign of benediction (ELLIOT, 1988; MCFARLANE, 2002).

Pragmatic ideas about surgical treatment date back to Henry CLINE, who performed the first fasciotomy in 1808 (GOSSET, 1968). The second fasciotomy known in medical history is attributed to COOPER (1824) (COOPER, 1824; VERHEYDEN, 1983; HUESTON, 1985).

Theodor KOCH (1897) deemed it obvious that mere severing of the aponeurosis could not result in an improved manual function. Because of the short term improvement, GOYRAN (1834) advocated total excision of the diseased fascial tissue.

But only progress in bacteriostasis and aseptic techniques fostered the development of more invasive surgical techniques. LEXER (1931) was—to our knowledge—the first to propagate complete aponeurectomy as well as dermatofasciectomy.

Despite more than 380 years of research, neither is the true pathogenesis of Dupuytren's disease understood, nor a causal therapy known.

Even for gaining a partial understanding of Dupuytren's contracture, a surgeon will have to enter into a topical discourse, which needs to include the basics of molecular biology and pathobiochemistry.

3 Epidemiology

The prevalence of Dupuytren's disease varies worldwide. The disease is common mainly in the western hemisphere, primarily among north Europeans or their descendants, as long as they stem primarily from the north European cultural area (Woolridge, 1988; Sennwald, 1990).

In the area once reigned by the Austro-Hungarian monarchy, Dupuytren's disease can be found as far south as the northern Adriatic Sea, a result of migration. The disorder is practically unknown in the southern Mediterranean and the Middle East (Brouet, 1986; Hueston, 1974, 1994, 1987).

Asians, African blacks of pure stock or Afro-Americans, native Americans (Indians), aboriginal Sami (Lapps) and Sinti and Romany peoples show only isolated cases of Dupuytren's (Zaworski et al., 1979; Plasse, 1979; Furnas, 1979; Maes, 1979; Rosenfeld et al., 1983; Ushijima et al., 1984; Egawa 1985; Mennen, 1986; Makhlouf et al., 1987; Richard-Kadio et al., 1990; Vathana et al., 1990; McGrouther, 1990; Leflore, 1991; Sladicka et al., 1996; Gonzalez et al., 1998; Finsen et al., 2002).

3.1 Prevalence of Dupuytren's disease among Caucasians

The prevalence of Dupuytren's disease in the unselected white North American population is about 1–2% (Bunnell, 1956; Conway et al., 1954; Gray, 1977; Crisp, 1984; Pal et al., 1987).

On the basis of one study, which examined 403 surgical and internal medical patients, Rafter et al. (1980) assessed the frequency of palmar fibromatosis among Irish people as 17% of those examined.

Mikkelsen (1972) conducted a screening program among 15,905 people in a Norwegian small town and found that 9.4% of men and 2.8% of women had Dupuytren's disease.

Mackenney (1983) examined 919 people in an orthopedic hospital in England and found that 5% of men and 3.5% of women were affected by the disorder.

3.2 The incidence of Dupuytren's disease in Germany

With respect to epidemiological data for Germany, we depend on the findings of Schnitzler (1935). He examined 80,000 surgical outpatients from Munich and found that 0.15% (n = 120 patients) had Dupuytren's disease.

Out of 985 unselected outpatients at the Department of Internal Medicine of the University Hospital in Mainz, 9.82% of men and 3.28% of women had flexion contractures (Knapp, 1987).

For the states of the former West Germany, no reliable data about Dupuytren's disease are available, whereas Beck (1954) and Lösch (1973) assume an incidence of 2.4% for the states of the former German Democratic Republic.

If these percentages are extrapolated to the total population of today's united Germany (79,670,000) (von Barata, 1991), a minimum of 1,354,390–1,904,113 (i.e. approximately 1.4 million to 1.9 million) Germans are affected by Dupuytren's contracture (Brenner, 1994; Brenner et al., 2001).

As the disorder implies a significant handicap as to the patient's work-life, the economic implications become obvious.

3.3 Age and Dupuytren's disease

Most publications provide evidence that there is a continuous increase in the prevalence of Dupuytren's disease with progressing age.

In young children, it is extremely rare to find histologically proven Dupuytren's disease (Dupuytren, 1832; Berger et al., 1985; Urban et al., 1996; Foucher et al., 2001).

Only five cases were reported in a study on 1 million American soldiers aged 21–30 years (Conway, 1954).

Among Australians (n = 3,711), Hueston (1962) found digitopalmar flexion contractures in 25.29% of men aged 60, but in only 20.4% of women in that age group.

In male Britons aged > 75 years, the frequency of Dupuytren's disease was found to be 18.1%, while it was merely 9% in women of that age group (Early, 1962).

The contracture can affect children and very old people, but it occurs more often in middle age (56.8 years). Among the north German men we examined, the highest incidence was in the sixth decade of life (31.3%), whereas in women the disease predominated in the seventh decade (39.4%) (Fig. 3a).

3.4 Sex distribution of Dupuytren's disease

It is not known yet why digitopalmar fibrosis affects women significantly less often than men. Holzrichter et al. (1982) named this phenomenon "androtropia". He found that this androtopia can be observed in all races and countries.

According to surgical data on German patients, the ratio of men to women is between 9:1 and 7:1, but the incidence increases to 3.7:1, parallel to womens' age (Skoog, 1948; Berge et al., 1988; Brenner, 1994). In northern Germany, the ratio is 2:1 for patients in their ninth decade of life. According to our studies (Fig. 3b; Brenner et al., 2001), only people nearly 100 years of age have the distribution of Dupuytren's disease described by Lane et al. (1988) et al.

In women, the peak manifestation occurs 15 years later than in men (Burch, 1966; Mikkelsen, 1972, 1990).

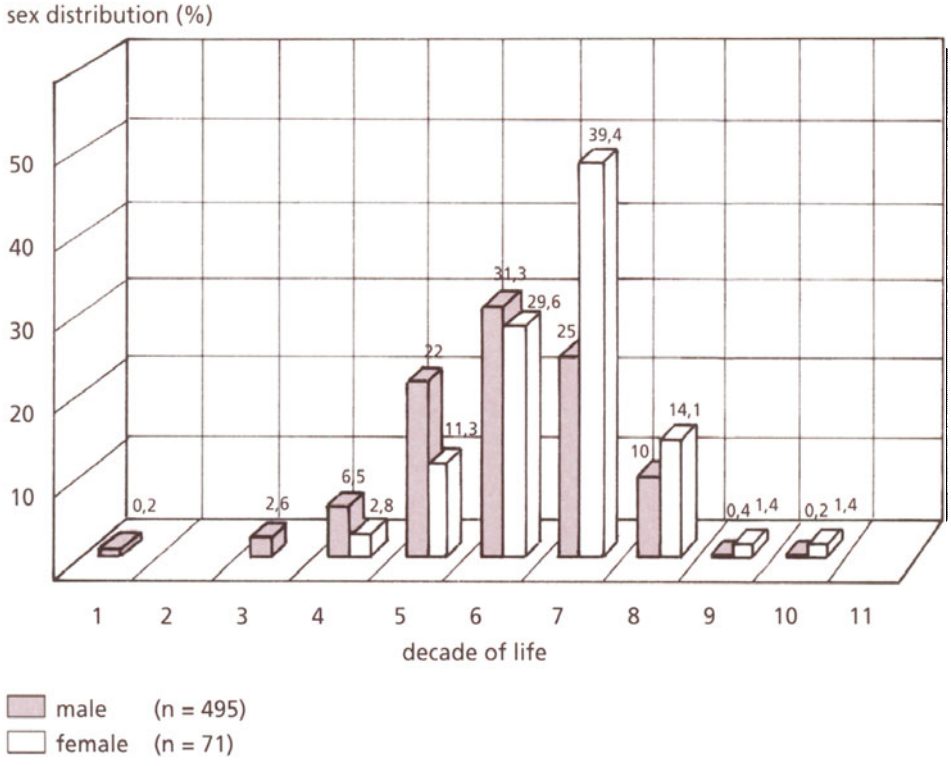


Fig. 3a. Age distribution by sex and decade of life. In men, the peak occurs between the age of 50 and 60 and in the 7th decade in women. Note the shift between the sexes (n = 566).

The hypothesis that gradual differences in the estrogen or progesterone receptors of the affected tissue cause the gender specific course of Dupuytren's disease could not be verified (HANKIN et al., 1986).

3.5 Handedness in digitopalmar contracture

Dupuytren's disease does not correlate with right- or left-handedness. Most frequently, flexion contracture starts unilaterally, but after sufficiently long observation intervals, the disorder will often affect the other hand. MILLESI (1965) observed bilateral Dupuytren's disease in about 81% of cases, and reported 12.2% exclusively right-handed and 6.5% exclusively left-handed cases.

According to MCFARLANE (1990), Dupuytren's disease affects both hands in two thirds of patients. SCHARIZER (1983) reports that both hands are involved in 81%, whereas only 55.1% of our patients had bilateral disease (BRENNER et al., 2001).

Data which seem to show that the right hand is more frequently affected, mainly relate to studies of patients who had undergone an operation. These data only prove that these patients were right-handed and had undergone fasciectomies on their dominant hand, as the disease had limited their daily activities.

COUCH (1938) and LING (1963) described identical frequencies of Dupuytren's disease in the hands of enzygotic twins, a fact not affected by occupation.

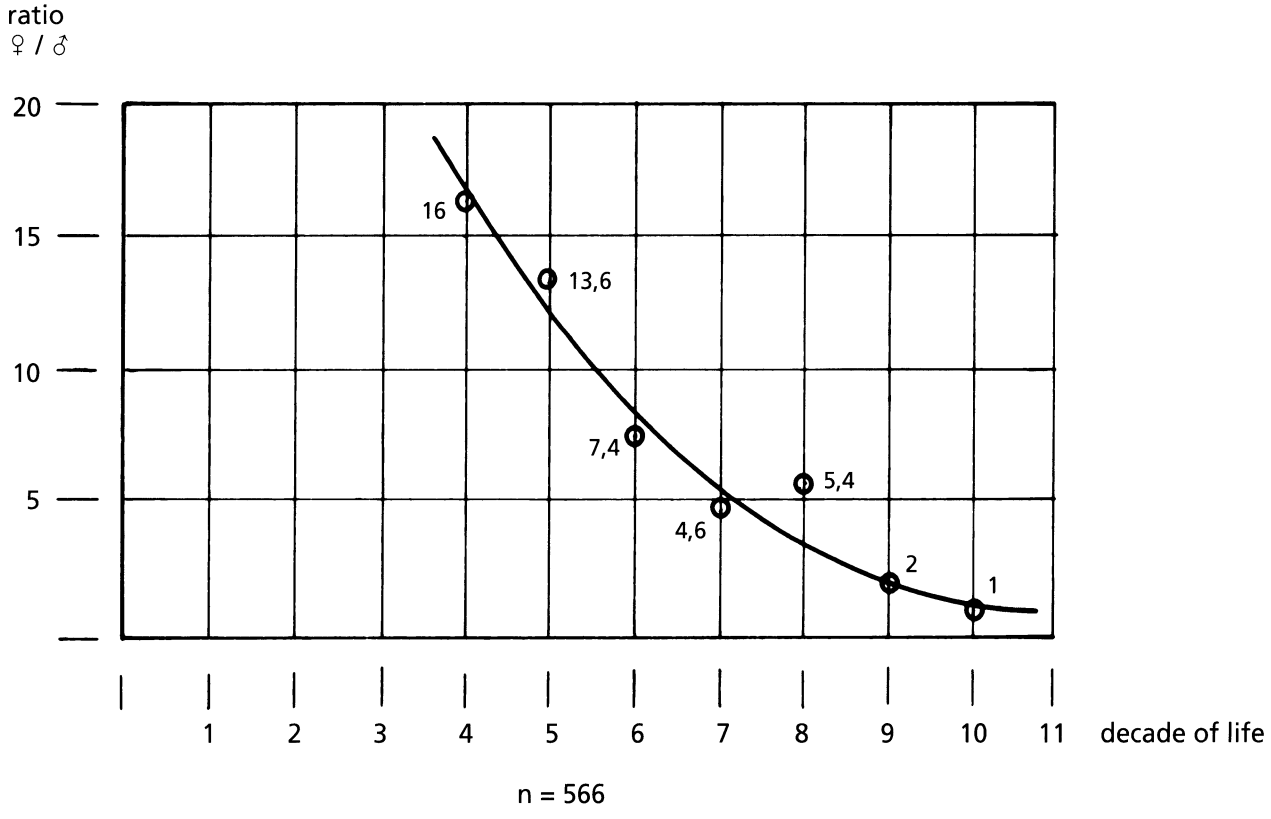


Fig. 3b. Equalization of the male/female ratio with increasing age: during the 4th decade the ratio is 1:16, during the 9th decade 1:2.

4 Theories concerning etiology and pathogenesis

The search for a link between Dupuytren's disease and other internal medical disorders aims at finding identical, reproducible pathways consistent with a common etiology. The many possible explanations that have been published in the literature cannot be reproduced here. Therefore, this book focuses on the most popular hypotheses and on those that are particularly controversial at the current time.

But even in the case of a statistically obvious association between an underlying disorder and Dupuytren's disease, it remains unclear whether both disorders have in fact common causes or whether the one merely influences the other (Manger et al., 1985; Köstler, 1987; McFarlane, 1988; Buck-Gramcko et al., 1992). Also, the coincidence of two or more disorders may be merely a function of a patient's age.

4.1 Association between trauma and Dupuytren's disease

The effort to find a link between chronic hand trauma and the triggering of digitopalmar contracture is as old as the medical history of Dupuytren's disease itself (Millesi 1967, 1981; Liss et al., 1996; Livingstone et al., 1999).

The ultimate reflection of this is the recognition of Dupuytren's disease as an occupational disease in some European countries (Bulgaria, Denmark, former Soviet Union).

In Anglo-American and German speaking areas, however, a link between repetitive trauma and the contracture is being dismissed mostly, and the process is therefore not recognized as an occupational disorder (Reichmann et al., 1968; Kirsch, 1975; Cotta, 1984; De La Caffiniere, 1986; Wulle, 1986).

The circumstance that Dupuytren's disease manifests itself with identical frequency in Australians and North Americans of Celtic-British origin (both groups) shows that environmental factors such as the type of occupation, various chronic traumata, and climate have hardly any influence on the development of the disorder (Lamb, 1981; Brenner 1994).

Although the incidence in office workers and those primarily engaged in manual activities is similar—and a strict and reliable differentiation shows semantic deficiencies—, cases of so-called microtrauma (tear-like interruption of longitudinal fibers) with proven deposits of hemosiderin due to previous bleeding have revived the discussion (Larsen et al., 1960; Skoog, 1967, 1970; Iwasaki, 1984).

We were able to prove, however, that the severity of Dupuytren's disease correlates with heavy manual labor. According to Tubiana's classification (expressed as a simple numerical score), the average score for the disorder in clerical workers was 3.17, whereas it was 4.21 in manual workers (Brenner et al., 2001). We are aware that in our society, which is characterized by increased leisure time, those with administrative or mainly non-manual jobs are still exposed to repetitive mechanical strain of the palm owing to excessive sports (such as golf, tennis, etc.).

Our figures exceed the data reported by Mikkelsen (1990), who published a score of 1.9 for intellectuals and 2.23 for those engaged in hard physical labor.

Cocco et al. (1987), Bovenzoni et al. (1994), and Liss et al. (1996) believe to have proof for a positive correlation between repetitive "vibrations" in people's occupational activities and connective tissue proliferation. This claim is based on epidemiological data and a meta-analysis.

The authors qualify their claim by stating that definitions of the terms "injury," "trauma," "man-

ual work" and "repetitive trauma" may cause semantic confusion. It may be assumed that this insecurity also applies to the term "vibrations."

MILLESI (1967) admits that undoubtedly there are isolated cases in whom a digitopalmar contracture may develop after a singular, acute traumatic injury, but such a coincidence is so rare that it seems unjustified to assume an etiological correlation (MILNER et al., 1985; STEWART et al., 1985; WYLOCK, 1989).

Sibyllinely, WILHELM (1971) concludes that a relation between trauma and fibromatosis can be assumed with sufficient probability if no changes of the Dupuytren type had been identified six months prior to the accident and an "inherited disposition" can be excluded.

4.2 Ectopic fibromatoses

Connective tissue deposits in other locations and occuring simultaneously with Dupuytren's disease, are known as ectopic fibromatoses. Such deposits are typically found on the second to the fifth digits or as plantar or penile fibromatoses. Deposits on the auricular concha, tongue, forearm, and Achilles' tendon are rare (MATEV, 1990; SINHA, 1997). Aggressive polyfibromatosis is the most extreme form, and it causes bilateral rigid flexion contractures of the hand as well as ubiquitous spontaneous keloid scarring (without prior trauma) all over the body (GONZALEZ-MARTINEZ et al., 1995; LEE et al., 1996).

4.2.1 Knuckle pads

The simultaneous occurrence of knuckle pads (Fig. 4) with digitopalmar fibromatosis is an argument in favor of the thesis that Dupuytren's disease is a systemic disorder (BOYES, 1969; LEGGE, 1985; GABIANI et al., 1986; MATEV, 1990).

Biochemically and histologically, ectopic fibromatoses correspond to palmar Dupuytren tissue (GABBIANI et al 1977; ADDISON, 1984; CAROLI et al., 1991; IRWIN et al., 1997).

Knuckle pads—previously referred to as "pulvillus digiti"—are regularly found on the dorsal side of the proximal interphalangeal joints of the fingers (RAMOS et al., 1956). LUND (1941) showed that the dorsal knuckle pads were known as "helodermia" early on, but the causal link to

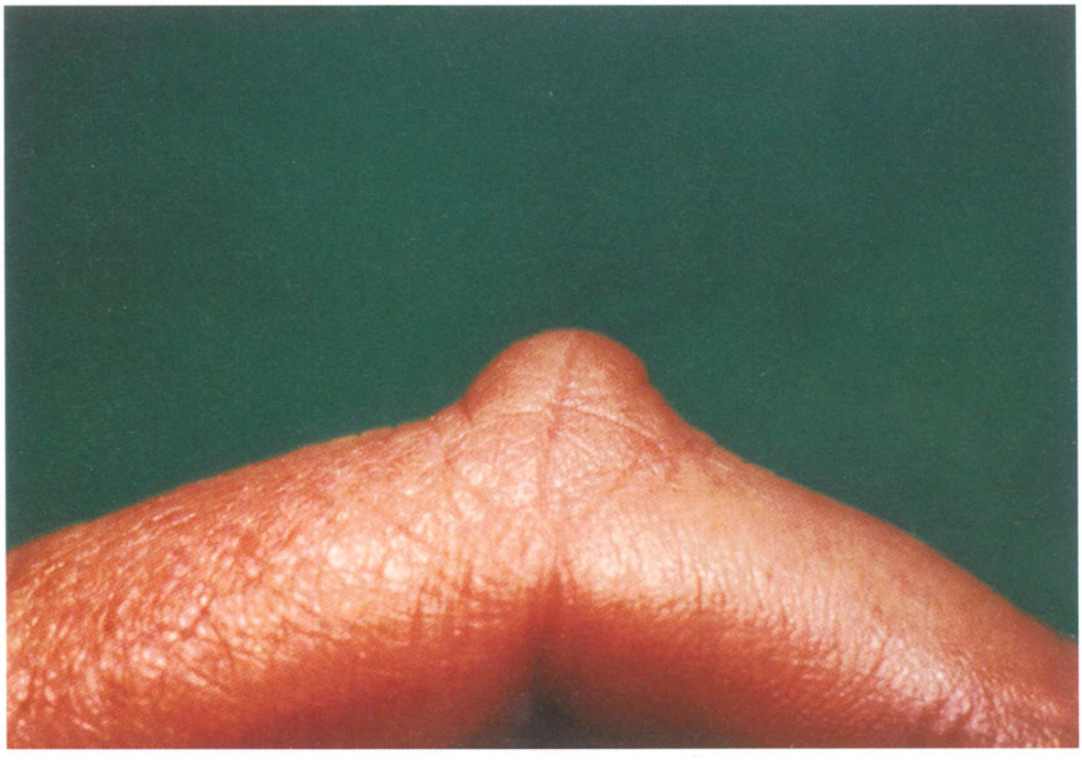


Fig. 4. Knuckle pads (Garrod's disease) are located dorsally above the proximal interphalangeal joints of the second to fifth digits.

Dupuytren's disease remained undiscovered for a long time. English language references associate knuckle pads with the name of GARROD (GARROD, 1893, 1904; MCINDOE, 1958; REICHERT et al., 1985; GONZALEZ et al., 1990; LASKIN et al., 1993; SINHA, 1997).

According to HUESTON (1963), the presence of knuckle pads allows the conclusion of a strong Dupuytren diathesis. WATSON et al. (1991) states that the knuckle pads are precursors of the actual flexion contracture and resolve spontaneously after complete expression of Dupuytren's disease. Our own observations do not tally with this view.

In the literature the reported frequency of knuckle pads varies between 11% and 44% in patients with Dupuytren's disease (SKOOG, 1948; BOYES, 1954; VIJANTO et al., 1973).

MCGROUTHER (1990) explains the diverging data with variable interpretations of morphological changes, whereby some authors regard already thickened skin as dorsal knuckle pads, but others regard only palpable nodules as knuckle pads.

ISELIN et al. (1988) distinguished between two variants of the dorsal Dupuytren's disease. Under the first variant, he subsumed all knuckle pads ("les coussinets dorsaux") that did neither interfere with flexion nor extension of the finger. Among the second group of dorsal Dupuytren's disease, he regularly found hyperextension of the distal interphalangeal joint from mechanical fibrotic contracture or from pathological involvement of the retinacular ligaments, which altered the range of motion. These were also the possible cause of a mediocre to poor functional result after surgery.

4.2.2 Plantar fibromatosis

Neither MADELUNG (1875, 1886)—who, in modern references, is incorrectly credited with having described the disorder first—nor LEDDERHOSE (1897), whose name has been repeatedly and accidentally associated with the disorder, but GERDY (1844) mentioned plantar fibromatosis first.

The reported prevalence of plantar fibromatosis (Fig. 5) among Dupuytren patients is 1.5–12%

Fig. 5. Plantar fibromatosis (Ledderhose's disease) with wavy, adherent skin in the instep area of the foot, and longitudinal cords. The weight-bearing parts of the foot are unaffected (heel pad, joint balls of the foot and lateral border of foot).

(MEYERDING, 1936; MILLESI, 1981; BROUET, 1986). ALLEN et al. (1955) and RAO et al. (1988) did not see a sex specific predominance, but according to DONATO et al. (1996), men develop plantar fibromatosis twice as often as women.

Immunohistologically and ultrastructurally, there is a complete analogy to Dupuytren's disease (DE PALMA et al., 1999). Although the lesions on the foot are identical, LEDDERHOSE'S disease can hardly be explained by heavy manual work.

Moreover, hardening of the plantar aponeurosis normally occurs in the non-weight bearing instep area of the sole (GORDON, 1964; HAEDICKE et al., 1989). Hammer toes related to fibrosis are recognized, but toe contractures caused by ectopic fibromatoses are rare (CLASSEN et al., 1992; DONATO et al., 1996).

4.2.3 Penile fibromatosis

In 1743 François DE LA PEYRONIE, physician to LOUIS XIV, reported on three patients with a painful erection and deviation of the penile corpus (WILHELM et al., 1971; HORTON et al., 1973; NYBERG et al., 1982; WOOLRIDGE, 1988). Since then, penile shaft fibrosis has been associated with the name of PEYRONIE.

Some 1% of the US hospital physicians examined have induratio penis plastica. A dorsal and a ventral variety of penile fibromatosis are distinguished (Fig. 6a and 6b).

Ten per cent of patients with Peyronie's also have concomitant digitopalmar contracture (JORDAN, 1998).

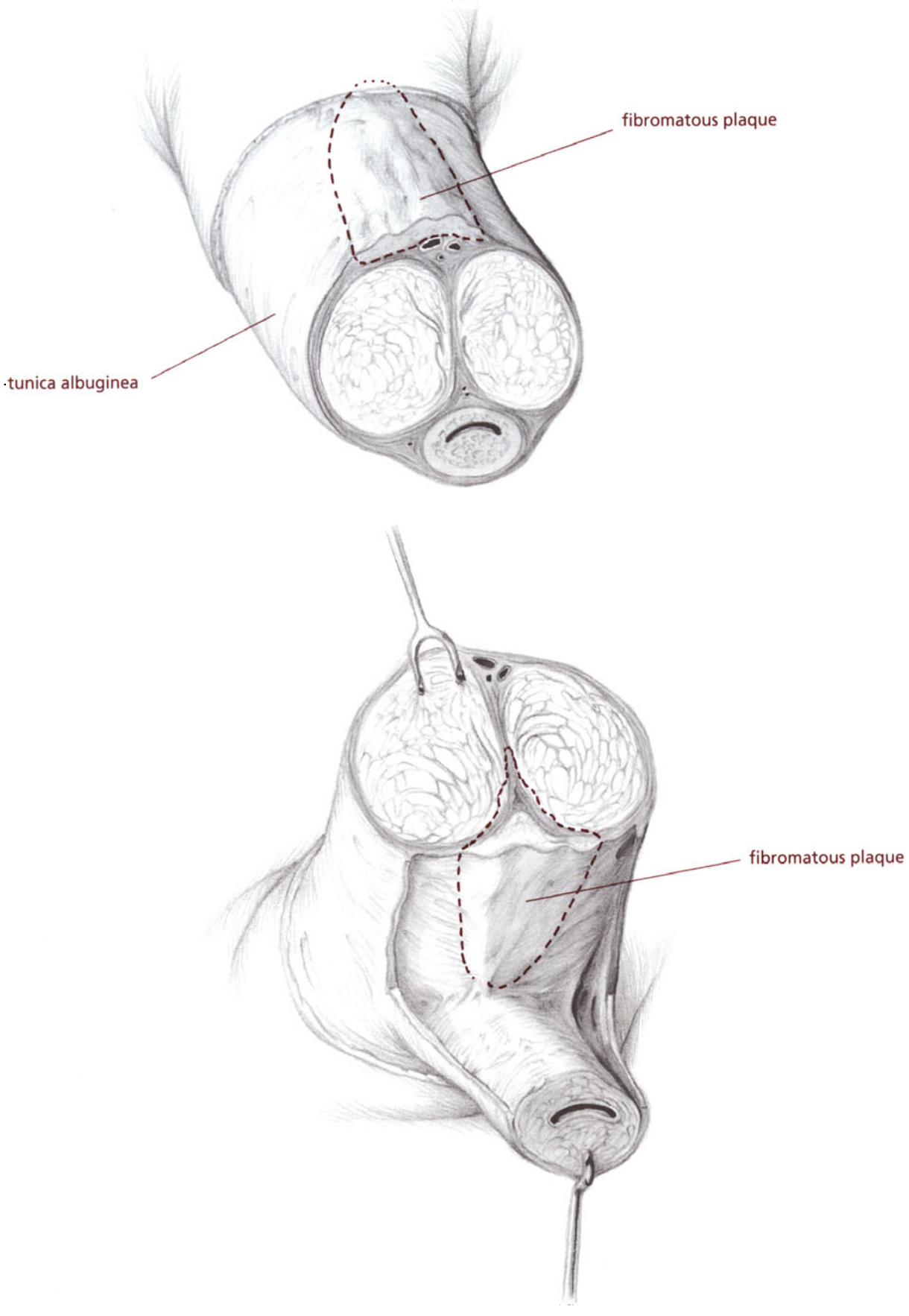
In our own series of 495 male Dupuytren's cases, we found penis plasticus in more than 0.9% of cases (BRENNER et al., 2001).

NYBERG et al. (1982) investigated penile fibromatosis among three families and confirmed an autosomal dominant trait in all three pedigrees. Seven out of eight examined individuals were HLA (histocompatibility antigen)-B-27 positive, which implies an pleiotropic effect of the same gene.

It is worth mentioning that the B7 cross-reacting group includes—apart from HLA-B7—B27, Bw22, B40, and Bw42.

Fig. 6a. Penile fibromatosis (Peyronie's disease). Dorsal type with connective tissue deposit at the back in the tunica albuginea of the corpora cavernosa. This fibrous accumulation "tethers" erection. In dorsal type of Peyronie's disease, the shaft is bent upward. Functional disturbance is only manifested if the deviation of the corporal body exceeds the "critical" value of 30 degrees.

Fig. 6b. Ventral type of penile fibromatosis. True Peyronie's disease is always associated with fibromatous plaques, which insert in the midline of the septal fibers of the center of the penis.


fibromatous plaque
tunica albuginea
fibromatous plaque

4.3 Hepatic cirrhosis and Dupuytren's disease

With respect to a possible link between Dupuytren's disease, chronic liver disease, and alcohol consumption, ATTALI et al. (1987) investigated 432 French inpatients of a gastroenterology ward by multivariate analysis and divided them into five groups:

1 89 patients with alcoholic cirrhosis;
2 55 patients with non-cirrhotic, alcoholic liver disease;
3 46 patients with chronic alcoholism without liver disease;
4 68 patients with non-alcoholic, chronic liver disease;
5 control group: 174 patients in a gastroenterology ward.

The prevalence of Dupuytren's disease was significantly higher among patients with histologically verified cirrhotic or non-cirrhotic alcoholic liver disease (25.5%) compared to patients with non-alcoholic liver disease (6%). But the relationship did not significantly differ among alcoholics with and without liver disease. Patients presenting with Dupuytren's disease were notably older ($p < 0.001$) and had experienced significantly more hand injuries ($p < 0.1$).

ATTALI et al. (1987) distinguished a total of nine variables which predispose to digitopalmar fibromatosis. In decreasing order these factors are:

1 age;
2 gender (male);
3 manual labor;
4 previous hand injuries;
5 diabetes mellitus;
6 daily alcohol consumption;
7 duration of alcohol misuse;
8 total alcohol consumption;
9 duration of nicotine abuse.

NOBLE et al. (1992) tried to find out if liver disease or alcohol is primarily responsible for the higher incidence of Dupuytren's contracture among these patients at risk. The authors investigated four different patient groups: Group A included 100 members of Alcoholics Anonymous in Manchester (United Kingdom), group B 82 non-alcoholic patients with posthepatic syndrome or primary biliary cirrhosis. Group C consisted of 50 patients from Cairo (Egypt) with bilharzia. None of these patients had flexion contracture. Control group D was recruited from age- and sex-matched trauma patients from a hospital in Manchester.

The prevalence of Dupuytren's disease was higher in the two group of patients with alcoholism (28%) and in the group of patients with non-alcoholic liver disease (22%) compared with the control group (8%). However, these differences reach marginally statistical significance ($p < 0.05$).

NOBLE et al. (1992) confirmed again that the frequency of Dupuytren's disease is probably higher among heavy drinkers, and that this effect is mainly secondary to liver disease induced by alcohol abuse, but that genetic factors have an even higher etiological impact.

4.4 Alcohol abuse

So far, data about the correlation between alcohol and Dupuytren's disease have often been ambivalent. According to HURST et al. (1990), the proportions of people who drink alcohol on a regular basis were almost identical in the control group (35%) and in the group of Dupuytren patients (39%), but in our study, the proportion of habitual drinkers with digitopalmar contracture reached a mere 14.7% (BRENNER et al., 2001); 53% of them suffered from bilateral Dupuytren's contractures.

BARLOW et al. (1986) rated 23% of their patients as heavy drinkers.

Because of the known difference between reported and actual alcohol consumption, BRADLOW and MOWAT (1986) conducted a validated, score adapted study. Alcohol misuse was defined as a daily alcohol intake in excess of 40 g. Two of a total of 10 patients with Dupuytren's disease, but none of the 44 female controls, consumed ≥ 40 g of alcohol per day. The alcohol consumption score correlated with urate and gamma GT (glutamyl transferase) serum concentrations. These data imply a link between alcohol consumption and the clinical course of Dupuytren's disease.

BURGE et al. (1997) based their prospective study for the Oxford region (United Kingdom) on the World Health Organization (WHO) AUDIT (Alcohol Use Disorders Identification Test), and delivered convincing evidence that in the group with Dupuytren's disease, weekly alcohol consumption (mean 7.3 units per week), significantly

exceeded consumption in the control group (5.4 units per week).

RABINOWITZ et al. (1983) found that alcohol causes incomplete fatty acid oxydation in location-matched digitofascial specimens, as reflected by higher octanoate concentrations and the accumulation of other short chain fatty acids. In combination, these two agents are responsible for subsequent hypoxia of the liver.

In addition, patients with Dupuytren's disease were often found to have pathological tissue concentrations of methyl esters and free cholesterol. These markers of lipogenetic activity seem to be indicators of mild hypoxia. Altered fat acts as an irritant, stimulating the proliferation of fibroblasts responsible for hepatic cirrhosis and also for the development of digitopalmar fibromatosis.

4.5 Nicotine abuse

Locally altered microcirculation and the generation of oxygen free radicals are being considered pathogenetic factors in alcohol toxic Dupuytren's disease and may explain the link between nicotine abuse and flexion contracture:

Among the patients in our Dupuytren study, 35.5% were smokers (BRENNER et al., 2001). This is a fairly low figure when compared to the strikingly high percentage of 68.2% smokers among AN's (1988) and other authors' Dupuytren patients. BURGE et al. (1997) proved that the mean lifetime cigarette consumption ("pack-years") was 16.7 pack-years in 22% of those with a digitopalmar contracture but only 12.0 pack-years in controls. These results are statistically significant.

LATHA et al. showed as early as in 1991, how cigarette smoking affects the tissue concentration of various glycosaminoglycan (GAG) fractions in animals. The GAGs are of particular importance in the etiopathogenesis of Dupuytren's disease as a systemic disorder of the connective tissue, as will be shown later in this book (BRENNER, 1994).

4.6 Epilepsy

Male patients with epilepsy are five times more likely to develop Dupuytren's disease than the population without epilepsy. In women this risk is 11-fold (EARLY, 1962).

With regard to the coincidence between epilepsy and palmar fibrosis, two hypotheses prevail:

1. Both epilepsy and Dupuytren's contracture are hereditary disorders with genetic causes.
2. The increased rate of digitopalmar fibromatosis in epileptics is a side-effect of their medication.

The value of the first hypothesis is supported by the fact that Dupuytren's contracture is often associated with idiopathic, and to a lesser degree to posttraumatic epilepsy. STUHLER et al. (1977) showed this in patients from Lower Saxony/Germany.

Just as in the population without epilepsy, there is an age-related increase in the incidence of Dupuytren's disease among patients with epilepsy. The disease incidence increased also with the duration of epilepsy (HURST et al., 1990).

As far as we know, no prospective study has ever proven a correlation between the intake of phenobarbiturate and the development of digitopalmar fibromatosis proven with statistical significance (LUND, 1941; ARIEF et al., 1956; HUESTON, 1982).

FRÖSCHER and HOFFMANN (1983) presented exemplary data on this subject. Their data explain the regional situation in Germany as well. The authors determined that 5% of all patients attending the outpatient department for epilepsy of the University Hospital in Bonn had both Dupuytren's contracture and epilepsy.

So far, we cannot provide a conclusive explanation as to why the prevalence of Dupuytren's contracture is significantly increased in epileptics.

4.7 The hypothesis of a neurogenic origin of Dupuytren's disease

The theory of a neurogenic origin of Dupuytren's disease is based on the observation that the ulnar types dominate clinically, and that there are some reports of ulnar nerve impairments (COTTA, 1984; SALZBERG et al., 1987). Cases of combined median and ulnar nerve irritations have been described in conjunction with Dupuytren's contracture, associated with a prolonged latency of sensory nerve action potentials (SNAP) and simultaneous electro-neurographical signs of a polyneuropathy (MUMENTHALER, 1970; MUMENTHALER and SCHLIACK, 1982). From a surgical point of view,

this seems to confound the principle of cause and effect. MILLESI (1959) pointed out that Dupuytren's contracture may very well affect areas beyond the fourth and fifth finger rays, which are supplied by the ulnar nerve. It is just a question of time until the fingers beyond the region supplied by the ulnar nerve will be involved.

4.8 Hereditary factors

JENTSCH (1937) reports on the concordant occurrence of digitopalmar contracture in a pair of male homozygous twins, which, in his opinion, was incontrovertible proof that Dupuytren's disease is an inherited disorder.

Current thinking is that digitopalmar fibrosis is inherited via a single gene (mutant allel) as a autosomal dominant disorder with variable penetrance (MCFARLANE, 1997).

A gene is dominant if it results in the same phenotype in a homozygous as well as heterozygous state.

The complete genetic and clinical expression in men occurs only after the 75th year of life. For women, the penetrance is lower. An exception are women with homozygosy from both parents (LING, 1963). Homozygosy means that the maternal and paternal genetic loci on the same autosomes are identical. If the maternal and paternal germ cells are different with regard to two complementary genes, the individual is heterozygous.

WELSH et al. (1990) point out that digitopalmar fibromatosis can by no means be explained by a sex-linked mode of transmission alone.

The gene in question is dominant, but only homozygosy induces an aggressive course of Dupuytren's disease with ectopic deposits or widespread disease.

Fig. 7. Example of the autosomal dominant transmission of Dupuytren's disease: a 35 year-old father and his 8 year-old son both suffer from an expansive form of digitopalmar fibromatosis, which extends from the ray of the thumb to the little finger.

In principle, all male and female carriers of the gene would develop Dupuytren's disease if they lived long enough.

Moreover, epidemiological studies on ethnic groups of pure stock demonstrate vividly that sporadic and spontaneous cases of Dupuytren's disease may even occur among children (Fig. 7). According to the literature, a new or "spontaneous" autosomal dominant mutation at any given genetic locus can be expected in about 1 of 100,000 newborns.

BOWSER-RILEY et al. (1975), SERGOVICH et al. (1883) and DE WEVER et al. (2000) showed by karyotyping that chromosomal mosaicism is frequent among patients with Dupuytren's disease.

By cytogenetic and molecular cytogenetic analysis DAL CIN et al. (1999) found a significant increase of clones in patients with Dupuytren's disease. The aberrations consist of a loss of the chromosome –Y and trisomies of the chromosomes +7, and +8.

Trisomy 7 is known from solid tumors and from normal brain and renal tissue, but trisomy 8 is always associated with fibrous tumors, for example, desmoid tumors. Trisomy 8 is also an indicator for the probability of a relapse in digitopalmar contracture.

4.9 Dupuytren diathesis

HUESTON (1963, 1984) coined the term diathesis for patients with Dupuytren's disease with a multitude of predisposing factors. The true Dupuytren's diathesis includes a strong family history, onset at an early age, occurrence of multiple and ectopic fibromatous deposits (Ledderhose's disease, knuckle pads, Peyronie's disease [WHEELER et al., 1981; HILL, 1985, 1989; MCFARLANE, 1990; TUBIANA, 1999]).

4.10 Coincidence of Dupuytren's disease and disorders of the immune system

4.10.1 Autoimmune disorders

Autoimmune disorders are characterized by a loss of the physiological immune tolerance (developed during fetal life) to the body's own tissues, resulting in a breakdown of the humoral and cellular immune system so that antigens cross-react and the immune system looses the ability to recognize autologous tissues as such. This type of immune response is pathologic and leads to the signs and symptoms of autoimmune disease. Autoimmunological aggression is therefore a false "self-recognition" and induces a poorly controlled, repetitive response to tissue injury or physiological degradation (BUSSE et al., 1983; JOSZA et al., 1988).

In 1959 MILLESI showed that tuberculoid reactions could be provoked at the injection site through autologous and homologous homogenized Dupuytren tissue harvested during surgery (immunization). Collagen was credited with having an antigenic booster effect.

BURCH et al. (1966) concluded from statistical studies that Dupuytren's disease might be an autoimmune disorder and saw the main cause for the disease in "forbidden clones of lymphocytes."

Analogously, this working hypothesis found its way into the literature as "clonal selection theory" (NAGAY, 1985).

GAY et al. (1972) also suspected that Dupuytren's disease is an autoimmune response. But it took until 1979 that MENZEL gave a statistically significant proof of serum antibodies to collagen type III in some patients with Dupuytren's disease. However, in seven of 24 patients a low concentration of antibodies to denatured type I collagen was found. PEREIRA et al. (1986) confirmed prior investigations: They tested sera from 16 Dupuytren's patients for IgG and IgM antibodies to native and denatured human collagen types I to IV. IgG antibodies to at least one of the collagen types were found in 69% of Dupuytren's patients, but only in 28% of blood donor controls. The authors explains the increased concentration of native type III collagen with an increased new type III collagen production; discretely raised incidence of type I and V collagen presumably reflects a non-specific cross-reaction. The elevated prevalence of antibodies to denatured type II collagen was interpreted as an indicator of a connective tissue disorder, such as rheumatoid arthritis. No HLA-DR4 increase was noted, however.

Since it was assumed that a T-cell mediated autoimmune disorder may result in the inflammatory process thought to be a causative factor for Dupuytren's disease, GUDMUNDSSON et al. (1998)

investigated the behavior of lymphocytes in 21 Dupuytren's patients versus 10 healthy blood donors. Dupuytren's patients had a marked increase in DR+ T-cells compared with the healthy controls. Even higher was the proportion of DR+ cells in those Dupuytren patients with palmar and plantar disease compared with those who had only digitopalmar fibromatosis.

At the same time, all Dupuytren's patients had a lower percentage of CD+-B-cells, compared with the normal population. This finding, which was marginally significant for all patients with fibromatosis, was most obvious in the group of patients with Dupuytren's disease with ectopic occurrence.

Such results support previous assumptions of an immunological etiopathogenesis. One can probably conclude that activated peripheral blood T-lymphocytes and possibly also B-cells may be a factor in triggering Dupuytren's disease.

Moreover, the therapeutic response to steroids as well as to γ-interferon supports the aforementioned autoimmune hypothesis of Dupuytren's disease.

4.10.2 AIDS

In addition to "traditional" autoimmune disorders it is primarily acquired immunodeficiency syndromes that explain why the prevalence of Dupuytren's disease in the normal population notably exceeds that in people infected with HIV. BOWER et al. (1990) compared the incidence of fibrosis in 50 patients with HIV infection (CDC stage IV: n = 48; CDC stage II: n = 2) with 50 HIV negative men with venereal diseases. Eighteen HIV positive patients (36%) had concomitant Dupuytren's disease, nine even bilaterally. "Thickening" of the palmar aponeurosis was diagnosed in another six patients. None of the male controls suffering from sexually transmitted diseases presented with digitopalmar flexion contracture. There is evidence for an increased activity of oxygen free radicals in HIV infected individuals, suggesting a single induction mechanism for Dupuytren's disease and AIDS.

In contrast, FRENCH et al. (1990) found only three cases of digitopalmar flexion contracture among 50 HIV positive patients. One was associated with alcohol misuse, another with cutaneous Kaposi's sarcoma combined with chronic active hepatitis, and the third with cytomegalovirus retinitis plus a pneumocystis pneumoniae. The authors assume that the data on frequencies diverge because of the contrasting clinical CDC stages (JARKE, 1993) for the acquired immunodeficiency syndrome. How much HIV subtypes that are not apparent in the HIV-I-antibody test have an impact here, remains a to be studied.

It must be added that HIV infections occur predominantly in adolescents and young adults. One may therefore assume from epidemiological studies that the prevalence of Dupuytren's disease is close to zero in this very young group, a number which is significantly less than even FRENCH's 6 per cent. The only exception from this are sero negative Dupuytren's cases at an early age and with strong family diatheses, none of which were found in any of the patients above. AIDS patients actually appear to have an increased prevalance of Dupuytren's disease.

4.11 Dupuytren's disease in diabetics

The first effort to find a link between diabetes mellitus and Dupuytren's disease dates some 120 years back (ROSENBLOOM et al., 1996).

With regard to statistical data on diabetes mellitus and its relation to Dupuytren's disease, one fact is striking: the numbers will depend on whether one examines patients suffering from Dupuytren's disease who have diabetes, or diabetics who eventually develop Dupuytren's contracture (MACHTEY et al., 1988; SUKENIK et al., 1988; RUFFINO et al., 1989; MCFARLANE, 1990; ROSENBLOOM et al., 1996).

Surgical studies on the association between Dupuytren's contracture and diabetes are mainly based on a positive history of clinically evident diabetes, because the authors regard digitopalmar flexion contracture a surgical problem of the extremity (GELDMACHER, 1970, 1994). It is easy to ignore one essential fact: the majority of diabetics will never reach the stage of crippling digitopalmar flexion contractures and will therefore rarely need surgical intervention (FOSSATI et al., 1975; LAMB, 1981; MCFARLANE, 1987; HURST et al., 1990; WELSH, 1990; BRENNER, 1994). That Dupuytren's disease tends to have a "milder" expression and a more radial course in diabetics, has been empirically verified.

In our own study, we confirmed the findings of NOBEL (1992)—who found generally "more benign"

prognosis for flexion contractures in diabetics. With 8.2%, the correlation between Dupuytren's contracture and diabetes was increased compared with the normal population or data found in the literature. At 3, Tubiana's average stage of Dupuytren's disease was lower in diabetics than in our patients (3.63). Thirty-seven per cent of diabetes-associated Dupuytren's patients, who we examined by us, also had bilateral extension deficits (Brenner et al., 2001).

Furthermore, there were no gender-specific differences in the incidence of Dupuytren's disease compared to the normal population (Mikkelsen, 1972).

Several surgical statistics lack important details, such as glucose-tolerance tests, for uncovering "chemical", respectively latent diabetes. Studies published by internists have a similar problem: they tend to underestimate the prevalence of Dupuytren's contracture—mainly due to misinterpretation of the clinical symptoms of digitopalmar flexion contracture (Hurst et al., 1990).

According to Noble et al. (1984) and Rosenbloom et al. (1996), the true incidence of Dupuytren's contracture in diabetes mellitus approaches approximately 40 per cent.

Arkilla et al. (1997) showed a highly significant correlation between digitopalmar fibromatosis, age, and type 1 (insulin dependent) diabetes ($p < 0.001$). Contractures are always associated with macroalbuminuria in type 2 (diet-controlled) diabetics, independent from any time-related variables.

Bergaoui (1991) reported limited joint mobility (LJM) of the interphalangeal finger joints in 21% of diabetics without any fibrosis.

In diabetics presenting with simultaneous digitopalmar contracture, the frequency of LJM rose to 46.25%. This difference was highly significant ($p < 0.00003$).

These findings support the studies of Jennings et al. (1989) and Rosenblom et al. (1996), who state that LJM and Dupuytren's disease are also significantly correlated with diabetic retinopathy and neuropathy.

Hoopes et al. (1977) proved that enzymes related to glucose metabolism are significantly more active in palmar fascia affected by Dupuytren's disease than in healthy aponeurosis. They also found that lysosomal hydrolytic enzyme activity (acidic phosphatase, beta-glucuronidase, beta-galactosidase, and beta-glucosidase), which initiate the breakdown of glycogen, were elevated by a factor of five to ten in Dupuytren's tissue.

According to Rosenbloom et al. (1996), advanced glycation end products (AGE) are thought to form as a result of the nonenzymatic reaction of glucose with proteins (glucation) with rapid structural rearrangement, dehydration and condensation, especially in diabetics with associated Dupuytren's disease. The authors use this to explain not only the changes of the digitopalmar continuum, but also the higher prevalence of periarticular, osteoarthritic changes, limited joint mobility (LJM) and flexor tenosynovites among diabetics.

AGE are said to attract monocytes and macrophages, which posses high-affinity receptors as binding sites for AGE products. Macrophages supposedly release proinflammatory cytokines that coordinate further degradation and removal of senescent material as well as continued remodeling.

AGE protein receptors can be found on other cells, on spindle shaped fibroblasts, smooth muscle cells, and endothelial cells.

In conjunction with collagen, AGE are responsible for cross-linking and fibrillar stability.

As shown in animal experiments, these macromolecular agglomeration can be blocked by aminoguanidine. A speculative future approach would be the therapeutic use of AGE inhibitors.

These views are supported by Vracko's hypothesis (1974) that in diabetics lamellation (basal lamina thickening) is induced by repetitive necrosis of the pericytes. This results in regeneration in the direction of the lumen of the original basal membrane, with a progressive diameter decrease of the fascial microvessels.

4.12 Vascular disease

Bajanbeleg (1972) showed that the palmar aponeurosis is interwoven with perforating arteries and veins. These findings are supported by Schrader et al. (1997), among others. Ikeda and Umeda (1993) used scanning electron microscopy to show that in humans and monkeys uniform straight and coiled capillary loops exist only in the thenar eminence, the ball of the fingers and the digits. Subsequently Ikeda et al. (1993) conducted comparative three-dimensional angiographic

studies in primates and confirmed these findings. These studies are also consistent with earlier findings published by, for example, KECSKES et al. (1975). According to KECSKES and coworkers (1975), perforating vessels originate from the superficial palmar arch and course into the hollow of the palm from five directions: (1) the thenar eminence, (2) the hypothenar eminence, (3) the ball of the finger (monticuli), (4) the ulnar and radial border of the hand, and finally (5) as sagittal perforators directly to the surface. These results complement those of CONWAY and STARK (1954), who also described a hypovascular zone in the hollow of the palm ("trigonum palmare" = palmar triangle). It is thought that this nearly avascular palmar triangle is responsible for central skin necrosis of the palm following total fasciectomy.

This is contrasted by the findings of SCHRADER et al. (1997), who in intraoperative dissections with magnification consistently found six to nine bundles perforating the palmar aponeurosis, each with one artery, at least one vein, and one nerve.

Microvascular occlusions are said to be a causative factor in the development of Dupuytren's contracture (COMTET et al., 1986).

KRAFT, MILLESI, and BLÜMEL (1973) already showed decreased fibrinolytic activity, intraluminal fibrin thrombi, and a "suffusion of the vascular wall" by fibrin and its fission products in the advanced stages of Dupuytren's disease.

In Dupuytren's disease the lumina of the microvessels are clearly narrowed with thickened endothelial walls, and they contain circumferential layers of mononuclear, spindle-shaped cells with lamellar arrangement around the basal lamina (MURRELL, 1990).

Fibrous obliterative angiitis with narrowing of the vascular lumina of two to three times their normal caliber was consistently seen by COMTET et al. (1985) and BAUER et al. (1985) in histological studies on accidentally severed digital arteries of patients with Dupuytren's disease. VRACKO (1970) had already shown that capillary lumina in skeletal muscle and the number of concentric basal lamina layers (lamination) significantly increase in a cranial to caudal direction at several body levels. This increase is modulated by several factors, such as age, "oxygen poisoning", and diabetes mellitus.

The phenomenon of microvascular occlusion is apparently the result of an excessive number of endothelial cells bulging into the lumen. Pericytes surround the capillaries.

With respect to the origin of the Dupuytren cell, KISCHER and SPEER (1984) assume on the basis of transmission electron microscopic observations that pericapillary satellite cells (pericytes) may be a possible source of new fibroblasts, when stimulated by hypoxia. Luminal narrowing leads to microvascular occlusion and chronic, local tissue hypoxia, which facilitates the conversion of primarily fixed (non-migratory) fibroblasts into mobile and contractile myofibroblasts, a process that GABBIANI (1972, 1973) named "modulation." Under such conditions fibroblasts develop new morphological, biochemical and pharmacological features.

KISCHER's (1982, 1989) pluripotent, migrating satellite cells (presumably pericytes) may transform to fibroblasts and into myofibroblasts, as they are seen in digitopalmar contracture, into hypertrophic scars, or keloids.

Remarkable in this context is the longitudinal and parallel realignment of the occluded microvessels to the tension lines of the Dupuytren cords.

In agreement with VRACKO's (1974) hypothesis, MURRELL and coworkers (1989) are convinced that adverse conditions, such as chronic hypoxia, result in pericyte necrosis. Prolonged hypoxia would thus induce subsequent regeneration within the outer layers of the old basal lamina with partial or complete capillary occlusion, finally leading to fibroblast proliferation.

The aforementioned hypothesis offers an especially attractive explanation for the trigger-mechanism in Dupuytren's disease: The microvascular phenomenon acts as a unified pathway that explains why diabetic microangiopathy, chronic hand trauma, cigarette smoking, alcohol abuse, and hepatic cirrhosis may produce digitopalmar fibrosis (HILL, 1985; KISCHER et al. 1989).

4.13 Oxygen free radicals and induction of Dupuytren's disease

The primary mediator in Dupuytren's disease remains unknown, particularly the agent responsible for the release of fibroblast growth factor (FGF) from endothelial or perivascular cells, which leads to the proliferation of fibroblasts and finally myofibroblasts as an angiogenetic response.

Possible causative factors are special oxygen free radicals. Radicals are atoms or molecules with a non-paired electron in their outer orbit. Oxygen free radicals are atoms or molecules produced by univalent reduction of oxygen (ELSTNER et al., 1987; WHITE et al., 1990). The strong urge of free oxygen radicals to scavenge other electrons turns them into highly reactive substances in tissue.

An increased number of oxygen free radicals is found under hyperoxemic conditions(respiratory distress syndrome), prolonged tissue hypoxemia (microvascular occlusion, post-ischemia reperfusion syndrome), inflammatory processes (autoimmune disorders), radiation damage (antineoplastic effect of irradiation) and under the effect of specific toxins (drug-induced hepatotoxicity) (JOYCE, 1987). As a result of excess radical formation and/or weakening of the antioxidant protection systems, "oxidant stress" occurs.

Activated polymorphonuclear leucocytes, monocytes, and macrophages produce oxygen free radicals.

Physiologically this is desirable for the lysis of migrating cells or for raising endothelial permeability. Additionally, radicals induce the release of chemotactical plasma substances. Clusters of such cell formations, however, cause tissue toxicity, as, for example, in autoimmune diseases or cartilage destruction in rheumatoid arthritis.

Antioxidants—so called scavengers—such as vitamins A, C, or E (retinol, ascorbic acid, and tocepherol), or glutathione, cystein, lycopene (in tomatoes) and zeaxanthin (in corn) are capable of providing the additional electron that is needed to complete oxygen reduction, thereby eliminating the oxygen free radicals (DUTHIE et al., 1988, WAGNER et al., 1996).

Without oxygen free radicals being scavenged, the adenosine triphosphate dependent cell membrane will not function normally, causing cell death (apoptosis; WILUTZKY et al., 1998).

In hypoxic conditions the energy production is transformed from efficient oxidative phosphorylization to inefficient anaerobic glycolysis. Local ischemia causes ATP to be broken down into the purine bases hypoxanthine and xanthine; hypoxanthine dehydrogenase is converted to xanthine oxidase, releasing oxygen free radicals (O_2•, OH•) and H_2O_2. It should be emphasized that, apart from ischemia, alcohol facilitates this transformation, a fact that supports the idea of an association between alcohol misuse and Dupuytren's disease (RUSSEL et al., 1989; MURRELL et al., 1992).

In this context, it is remarkable that higher concentrations of hypoxanthine dehydrogenase are found in skeletal muscle and capillary endothelial cells than in other cells in the body. A reaction of hypoxanthine with molecular oxygen results in uric acid and free oxygen radicals. Either the resulting free radicals, whose half-life is only milliseconds, or the products resulting from their decay, harm the lumina of microvessels or can, at least in theory, make pericytes or fibroblasts change into myofibroblasts. This process accelerates narrowing of the microvascular lumina, which in itself perpetuates localized ischemia and the production of further free radicals (RUSSEL et al., 1989; MURRELL et al., 1994). Nicotine abuse also promotes this common pathophysiological response.

MURELL et al. (1987, 1990) showed convincingly that pathological fascial tissue taken from Dupuytren patients contains a concentration of hypoxanthine six times higher than that of apparently normal fascial tissue. If the latter reacts with hypoxanthine oxidase, further free oxygen radicals are produced. It is estimated that tourniquet application in healthy individuals increases the hypoxanthine concentration by 10%, whereas the global increase in patients with Dupuytren's disease is approximately 600%.

In palmar aponeurosis with Dupuytren's disease, hypoxanthine concentration increases with cell density and is two times greater in tissue classified as "nodule" than in "cord". The relation is 0.32 μmol hypoxanthine/g wet weight versus 0.14 μmol hypoxanthine/g wet weight ($p < 0.005$).

According to MURRELL et al. (1992, 1994), endogenously released radicals also stimulate the fibroblast proliferation (Fig. 8).

It should be mentioned that fibroblasts generate and release their own radicals, even in cell cultures (MURRELL et al., 1992).

Finally, TRELSTAD (1981) demonstrated that oxygen free radicals hydroxylate proline and lysine remnants nonenzymatically, and thereby increase collagen production and initiate angiogenesis.

When released into the extracellular matrix, oxygen free radicals cause tissue damage by lipid peroxydation, disruption of organelle membranes, and induce degradation of glycosaminoglycan hyaluronic acid (WHITE et al., 1990).

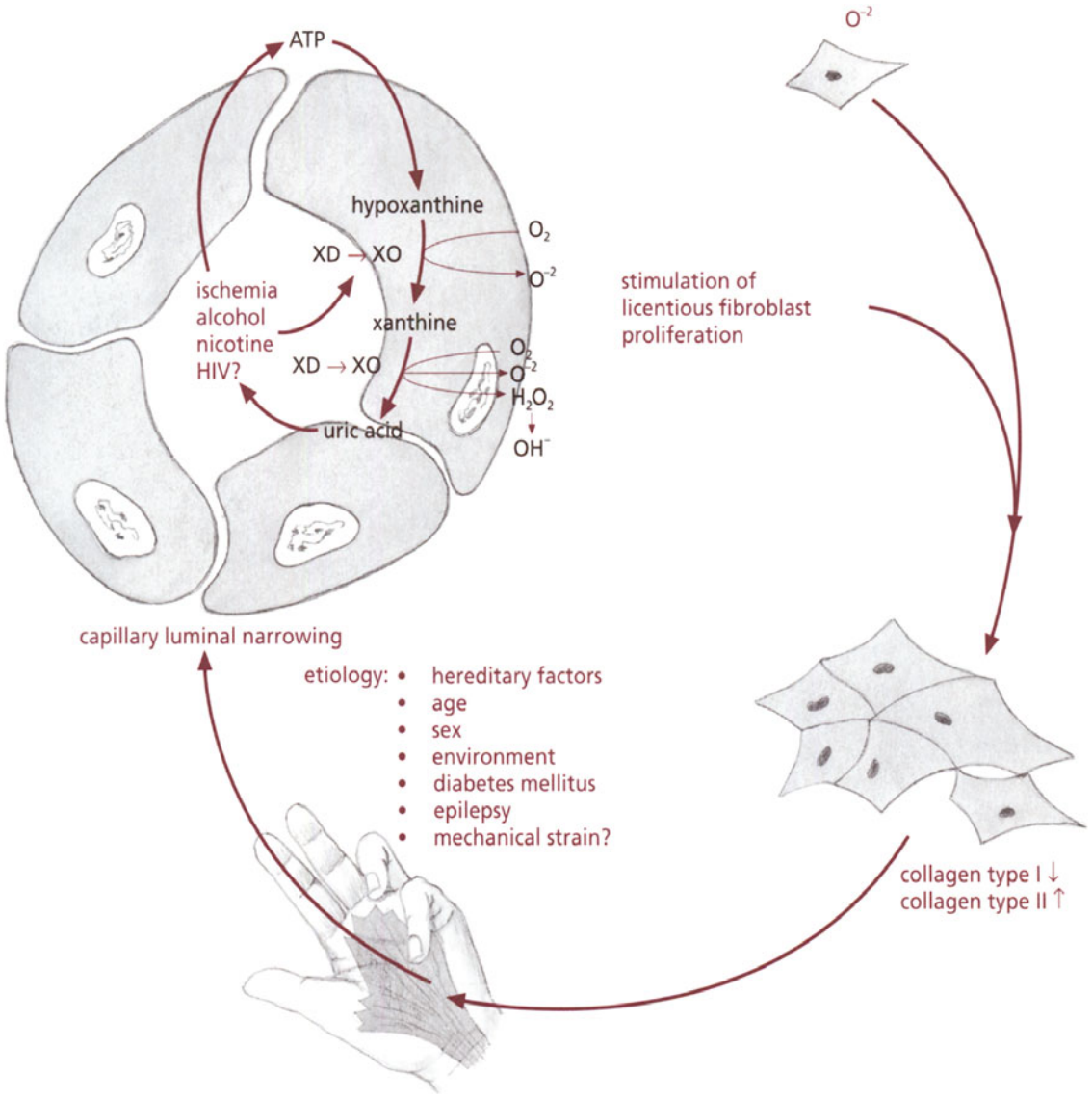


Fig. 8. Simplified explanation of the etiopathogenesis of Dupuytren's disease on the basis of the oxygen-free radicals theory: capillary luminal narrowing paired with co-factors, age, hereditary factors, environment, gender and endogenous factors such as diabetes mellitus and epilepsy, and possibly, mechanical strain, facilitate progressive ischemia. This is perpetuated by oxygen free radicals released under the influence of, for example, alcohol, nicotine and possibly also HIV infection. The efficient aerobic phosphorylization gives way to inefficient anaerobic glycolysis in the presence of oxygen-free radicals, and its end product is uric acid. This environment stimulates the proliferation of fibroblasts. At the same time, synthesis of collagen type I decreases and the formation of type III collagen increases, which shifts the balance.

5 Ground substance and cells of the extracellular matrix

5.1 The extracellular matrix

Connective tissue consists of cellular (10%) and extracellular (90%) components.

To explain its structure—which contains collagens and glycosaminoglycans as well as cells—the extracellular matrix can be compared with concrete (a mix of sand and cement with added pebbles) reinforced by steel (ordinary steel mesh). In analogy, the connective tissue cells correspond to the pebbles, the steel mesh to the collagen fibrils, and the sand-concrete mix to the glycosaminoglycans.

5.1.1 Mobile and fixed cells of the extracellular matrix

The cells of the extracellular matrix can be distinguished by their migration behavior. Fixed (stationary) cells include fibroblasts and fibrocytes, whereas histiocytes, mono-, and lymphocytes are mobile (wandering cells) (KRSTIC, 1988).

The most important, stationary connective tissue cell is the fibroblast with its pseudopodiae, which forms a lattice or mesh structure in which the mobile connective tissue cells move. The precursor of the fibrocyte is the spindle shaped fibroblast, one of the most multifaceted cell types known to man (MAINARDI, 1989; TOMASEK et al., 1999). The fibroblast is a biosynthetically active, cell, whereas fibrocytes are regarded as resting, biosynthetically inactive cells (Fig. 9a–9c).

Since GABBIANI and MAJNO (1972), myofibroblasts are regarded as the morphological hallmark of Dupuytren's disease. On the one hand, they contain a lot of mitochondria and rough endoplasmatic reticulum, which seems typical of metabolically active fibroblasts, but on the other hand they contain large bundles of actin microfilaments, which resemble the myofibrils of smooth muscle cells.

GABBIANI (1985, 1986) therefore called the myofibroblast a specialized contractile fibroblast.

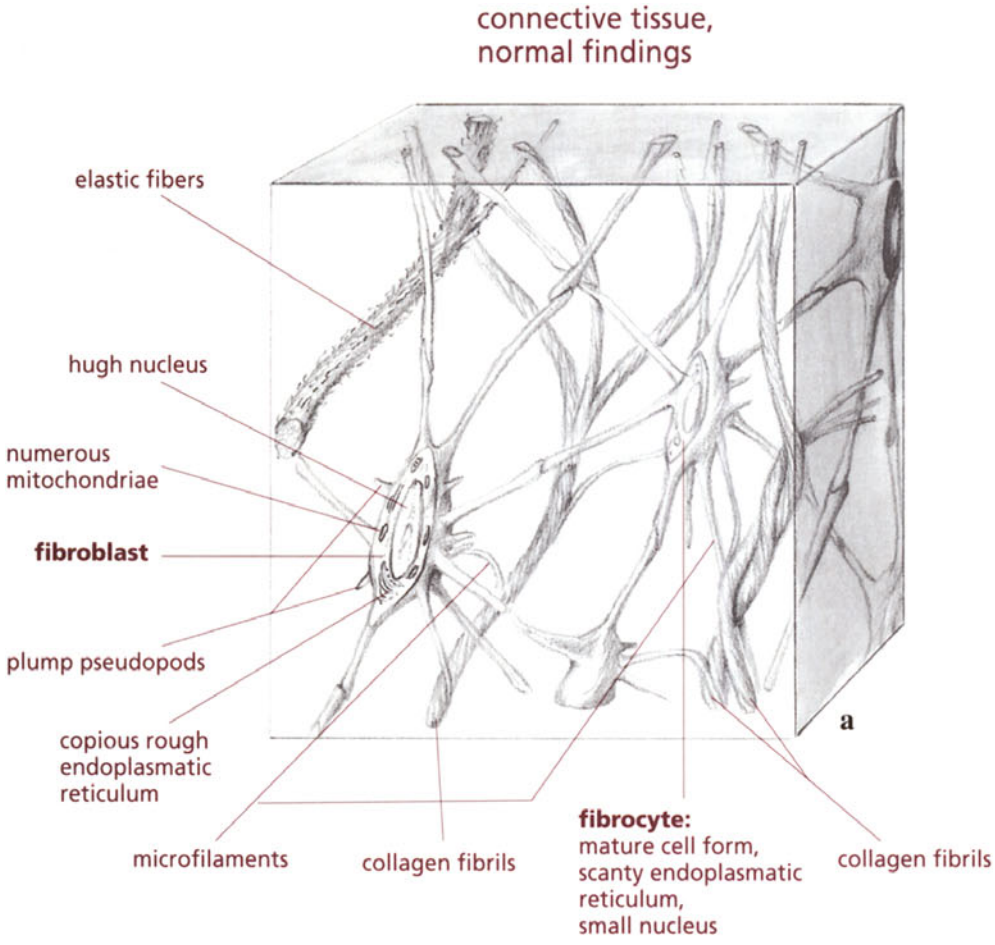
Its special ability to contract caused MAJNO (1979) to originally use the term "fibrospasts" for later stage tractoblasts. Because of the cell's many smooth muscle cell attributes and because the other term might be misinterpreted, the term myofibroblast became established.

The histogenesis of myofibroblasts remains unclear. Possible precursor cells are, among others, immature fibroblasts, monocytes, mesenchymal stem cells, and pericytes. Monoclonal antibody typing of the cytoskeletal composition may elucidate the cellular origin of myofibroblasts in tissues affected by Dupuytren's disease.

SKALLI's (1989) and SCHÜRCH's (1990) efforts to classify these cells based on four cytoskeletal forms showed the following characteristics. 1. Phenotype V: exclusively vimetin positive myofibroblasts. Vimetin positive cells seem to originate from fibroblasts. 2. Phenotype VAD: In this class, the myofibroblasts have positive receptors for vimetin, alpha-actin and desmin. Alpha-smooth muscle actin and desmin are indicators of smooth muscle attributes. Alternatively these cells can originate from pericytes. More desmin is found in non-muscle mesenchymal cells, such as endothelial cells. 3. Phenotype VA: with proof of vimetin, alpha-smooth muscle actin. 4. Phenotype VD: Myofibroblasts are positive for vimetin and desmin.

An argument against endothelial cells as the cellular origin is the negative reaction of monoclonal antibodies to von-Willebrandt-factor (SACHSE and BRENNER, 1994).

The connective tissue cells in Dupuytren's disease do not originate from leucocytes (anti leucocyte common antigen). On the basis of four macrophage antibodies (27E10 = indicator of acute tissue inflammation, RM3/1 = indicator of intermediary phase of inflammation [recovery phase], 25F9 = indicator of late inflammatory


connective tissue,
normal findings
elastic fibers
hugh nucleus
numerous
mitochondriae
fibroblast
plump pseudopods
copious rough
endoplasmatic
reticulum
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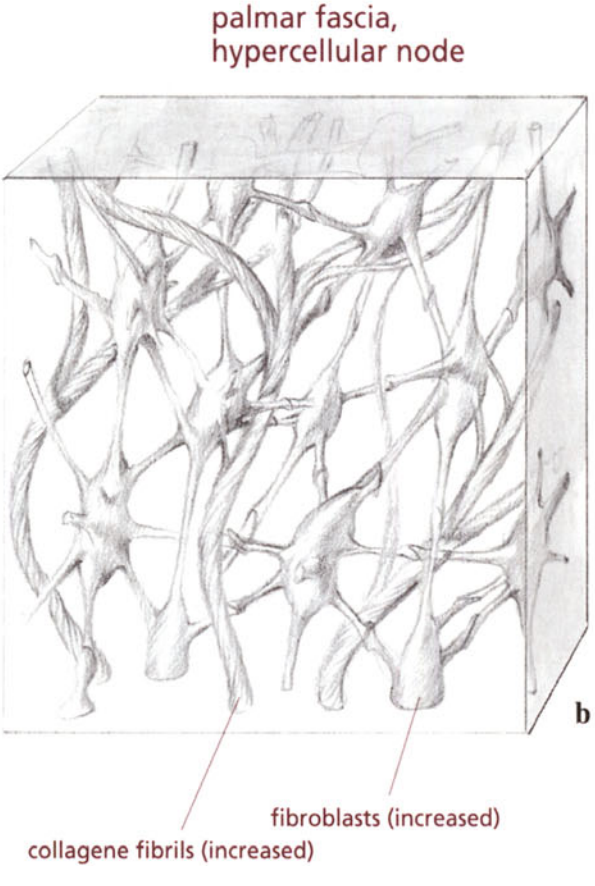

palmar fascia,
hypercellular node
b
fibroblasts (increased)
collagene fibrils (increased)

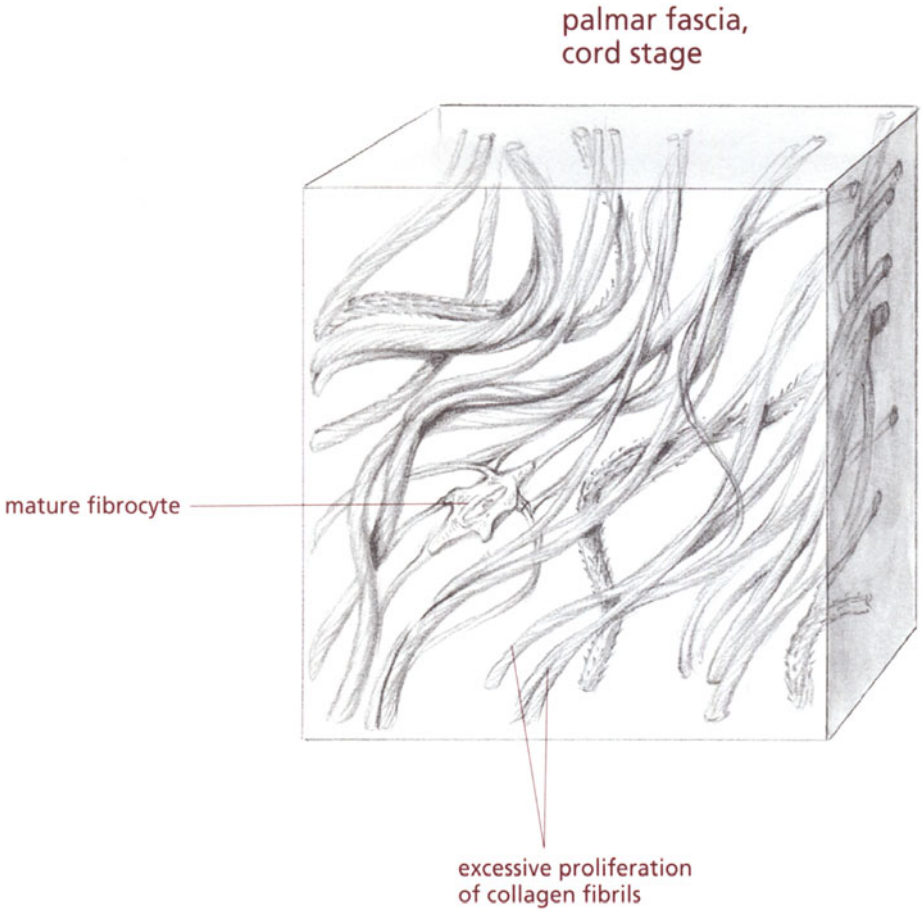


Fig. 9a. Connective tissue, normal findings. The long oval-to-spindle-shaped fibroblast (left) is the most versatile connective tissue cell. It has many plump pseudopods (micro spikes, lamillopodia). The copious rough endoplasmatic reticulum, mitochondriae and pinocytotic vesicles indicate that the cell exhibits a high rate of synthesis. The microfilaments important for contraction extend in an intra- to extracellular direction. They extend from the cell to the collagen fibrils and continue within the collagen bundles. The cytoplasm of mature (inactive) fibrocytes is sparse and the endoplasmatic reticulum is scanty, owing to its scarce synthesis. The tree root-like structures are the elastic fibers. The spaces within this mesh are filled by glycosaminoglycans. Their water-retaining properties create a jelly-like sol.

Fig. 9b. Palmar fascia: hypercellular node.

phase, CD68 = indicator of human, mature tissue macrophages) we showed that Dupuytren tissue contains myofibroblasts of varying developmental stages, which are of an "inflammatory" nature. With respect to their morphological surface antigens, macrophage mimicry is evident (BRENNER et al., 1996).

AZZARONE et al. (1983) showed that cells cultured from Dupuytren nodules represent an intermediary stage between normal human adult and virus-transformed fibroblasts.

But cells with the antigenic properties of macrophages can imitate their synthesis and release properties.

Even the "burnt out" cord stage shows tissue macrophages of the late inflammatory phase.

In the cord stage (TUBIANA stage IV) no receptors for epidermal growth factor receptor (EGFR) can be found, making the "external theory"—the genesis of Dupuytren's from skin fibroblasts—unlikely. According to HUESTON'S (1963) external theory, the supposed primary lesion lies in the fibrofatty tissue and the connective tissue skeleton is involved secondarily.

Excess growth factors and abnormal receptor expression can selectively stimulate cells as well as the extracellular matrix.

Basic fibroblast growth factor (bFGF), platelet derived growth factor (PDGF), and transforming growth factor subunit beta (TGF β), are all known to have effects on the proliferation of fibroblasts. Basic FGF stimulates fibroblasts and angiogenesis and binds the glycosaminoglycan heparin. In response to bFGF, Dupuytren's cells increase collagen production. Platelet derived growth factor (PDGF) has a mitogenic effect on in vitro cells of connective tissue origin (ALIOTO et al., 1994).

Apparently TGF β induces the differentiation of original fibroblasts into contractile myofibroblasts. In vitro studies have shown that intracellular TGF β 1 is found in fibroblasts, myofibroblasts, and capillary endothelial cells. During their proliferative and involutional stages, TGF β 2 is concentrated inside myofibroblasts, (BADALAMENTE et al., 1996).

LUCK'S (1959) widely accepted classification evaluates the proliferative changes of cells, distinguishing three stages. During the first stage, a focal fibroplasia exists. The fibroblasts proliferate because of a "purpose free arrangement." Typical for this proliferative stage is the accumulation of immature fibroblasts with myofibroblast mimicry. In their second stage, also called involutionary phase, the cells respond to the effective mechanical strain. The expression of PDGF through fibroblasts increases with increasing mechanical strain. Normal fibrocytes as well as fibrocytes from Dupuytren's disease express receptors for PDGF (ALMAN et al., 1995).

The residual third stage is mostly acellular: cord-like structures form. It coincides with shrinking of the connective tissue and clinical contracture.

The degree of contracture corresponds to the amount of lysophosphatic acid (LPA), released by activated platelets, or thrombin. It is remarkable that thrombin is only 70 per cent as effective as LPA (RAYAN et al., 1996; TOMASEK et al., 1999).

It should be stressed that the above-mentioned fibroblasts produce collagen and glycosaminoglycans, permanently altering the ground substance in which they reside. These changes of the extracellular matrix composition in turn influence the surface and secretion characteristics of fibroblasts. The terms used in this context are auto- or paracrine situation (QUAGLINO et al., 1997).

Growth factor TGF β can affect the amount of extracellular matrix by two mechanisms: (1) by increased synthesis; (2) by reduced decay. TGF β is an ambivalent growth factor that, on the one hand, stimulates the growth of mesenchymal cells such as fibroblasts, and, on the other hand, inhibits ectodermal cells such as keratinocytes or lymphocytes (BLOBE et al., 2000).

It is remarkable in this context that the TGF β receptors are also proteoglycans (KLOEN et al., 1995).

PDGF and epidermal growth factor (EGF) induce the proliferation of myofibroblasts. These proliferating fibroblasts are termed myofibroblasts. These specialized and proliferative fibroblasts are primarily located inside the Dupuytren nodules, where the further process originates (LUBAHN et al., 1999).

It should be added that EGF and TGF β belong to the same family and are stimulated by testosterone, which unfolds their angiogenetic potential (IBELGAUFTS, 1992).

5.2 Fibrous proteins

5.2.1 Structural proteins

Collagen is the best-known structural protein (Fig. 10).

One of the most striking biochemical characteristics of Dupuytren's disease is the progressive and irreversible accumulation of "excessive" fibromatous collagen.

The global collagen content corresponds approximately to the hydroxyproline content.

By light microscopy the collagen fibers lose their waviness and transparency. Overall they appear thickened and entangled with each other.

In the apparently unaffected palmar fascia, collagen concentrations are 20–25% higher than in healthy fascial tissue. The highest relative increase in collagen is found in the Dupuytren nodule (70%).

1 mg of dry tissue contains 60.4 µg collagen, apparently unaffected fascial tissue 73.4 µg; in "mildly" affected tissue the collagen concentration is 87.4 µg/mg, in longitudinal cords 91.3 µg/mg, and 108 µg/mg in the dry Dupuytren nodule.

There are at least 12 genetically distinct types of collagen in connective tissue, which are partly tissue or structure specific. Type I collagen is typical of normal tissue of the palmar aponeurosis; the proportion of type III collagen is below 3%.

In the Dupuytren nodule this proportion is as high as 10–15%, reaching 30–40% in the fibrous cords (Bailey, 1990; 1994).

In summary we observe the following biochemical alterations of the collagen metabolism in the palmar fascia affected by Dupuytren's disease:

(1) An increase in the functionally important residues of amino acid of hydroxylines, (2) hydroxynorleucine, a cross networking protein, stands out, and (3) an apparent increase in type III collagen in fascia with Dupuytren's disease to an average of 25% of the total tissue collagen content, and an almost vanishing proportion of type III in the normal aponeurosis (1–2%) as well as a slight increase in type V and type I collagen in superficial fibromatosis.

Murrell (1990) contradicts these results on the basis of his in vitro analyses. Fibroblasts cul-

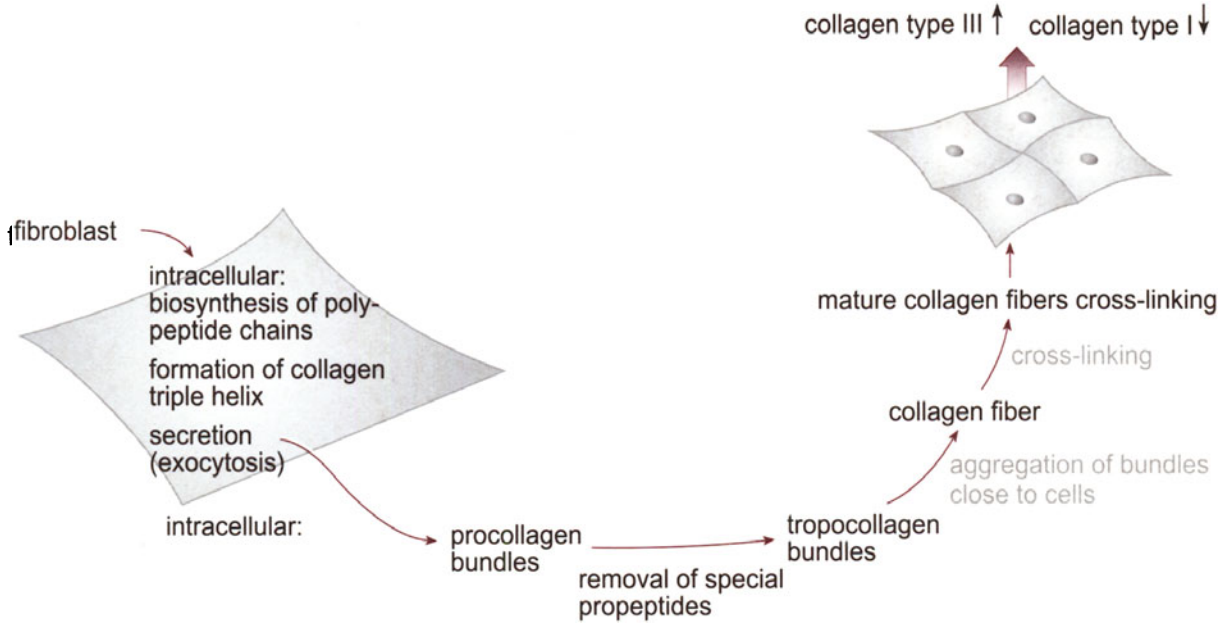


Fig. 10. Inside fibroblasts, many polypeptides are synthesized. These are needed for the formation of the triple helix. The intracellular products are actively transported to the outside (exocytosis). To achieve a higher tensile strength, these peptides are cross-linked to pro-α-chains and also assembled to procollagen bundles, which develop into tropocollagen ones. The precursor tropocollagen is converted to triple helices and finally to collagen molecules by the removal of special propeptides. Hydroxyproline and hydroxylysine are remnants that are rarely found in other proteins. Their detection is always an indirect parameter of the rate of collagen synthesis. Moreover, hydroxylysine and hydroxynorlysine are essentially absent in non-affected, healthy digitopalmar fascia. Collagen fibers aggregate in close proximity to cells. A high fibroblast density facilitates the synthesis of collagen type III, and the amount of type I collagen decreases simultaneously.

tured from healthy skin of the forearm, from apparently normal fascial tissue, and those from pathological Dupuytren tissue synthesize the same type of collagen. An intrinsic aberration of collagen production, for example secondary to genetic factors, seems unlikely. It is mainly local factors that change collagen metabolism. The increase in the ratio of collagen III to collagen I coincides with an increase in the density of cellular fibroblasts. Concomitantly, this changing ratio precedes a secondary inhibition of the production of collagen I, whereas the synthesis of type III is not impaired by the cell density of fibroblasts.

This is also true for fibroblasts from healthy skin, hypertrophic scars, keloids, tissue samples from the flexor retinaculum, and fetal skin.

It has remained unclear, however, why the cell density of fibroblasts influences the production of type I collagen.

If the high cell density hinders fibroblasts from making full use of their capacity to synthesize collagen type I, a shift in the ratio between type I and type II results. MURRELL therefore concludes that the fibroblast metabolism in Dupuytren's disease is not pathological.

These findings supplement the studies by SLACK et al. (1982), who described reproducible changes in glycosaminoglycans with different cell densities in fibroblast cultures.

5.2.2 Adhesive proteins

The adhesion sites of myofibroblasts described by RYAN et al. (1974), which he initially termed "microtendons," were rediscovered as "adhesive proteins" and rechristened "fibronexus" (MAJNO, 1979). By means of these attachments myofibroblasts exert a pulling action on the stroma.

Fibronectin is the best characterized adhesive and extracellular glycoprotein, and it is also known as cell surface protein or large, external, transformation-sensitive (LETS) protein.

The metabolism of the various isomers of fibronectin is concentrated on the myofibroblast inside the active and proliferating Dupuytren's nodule. ED + B fibronectin synthesis is a characteristic feature of the myofibroblast (BERNDT et al., 1995, 1998). Fibronectin concentrations remain high during the involutional and contractile stages, where as the disease progresses from the evolutional to the residual stage, the fibronectin level decreases gradually (HALLIDAY et al., 1994).

Fibronectin's main role in Dupuytren's disease is the adhesion of fibroblasts to surrounding type I and type III collagen. Additionally, fibronectin needs to transmit the intercellular contraction to the extracellular matrix (TOMASEK et al., 1987; BERNDT, 1994). Fibronectin is thus important for remodeling processes of the connective tissue (HALLIDAY et al., 1994). Transforming growth factor (TGF) β-1 stimulates the fibronectin synthesis and its receptor; it plays a vital role in the regulation of fibronectin and its "adhesive attributes" in Dupuytren's disease (TOMASEK et al., 1999).

A highly organized layer of extracellular material, the so-called basal lamina, always surrounds smooth muscle cells. The main component of this is laminin, which facilitates the adhesion of smooth muscle cells to type IV and other types of collagen, to heparin, and to heparan sulfate (both are glycosaminoglycans). Laminin also helps in selective compartmentalization and acts as a filter for macromolecules. Because fine fibrils (3–5 nanometers) are interwoven with excessively large fibrils (10–13 nanometers), the basal lamina would more appropriately be called filamentous, extracellular material (TOMASEK et al., 1987; YURCHENCHO, 1989).

Desmin is a global marker of muscle differentiation and it can already be detected in the early stages of embryogenesis. It does, however, not allow definite differentiation between the diverse types of muscle.

Apart from desmin, vimetin is also a type II-IF protein. It is characteristically found in cells of mesenchymal origin, such as fibroblasts, endothelial cells of blood vessels, and primarily in leucocytes. Newly cultured cells often express vimetin (ALBERTS et al., 1990).

The presence of intracellular microfilaments, also called cytoskeletal proteins (BRANDES et al., 1996), is an essential characteristic of myofibroblasts. Microfilaments consist of myosin filaments as well as actin and other "associated" proteins. There are five isoforms of actin (alpha-skeletal and alpha-cardiac, alpha-smooth muscle and gamma-smooth muscle actin, and the beta and gamma forms as non-muscle actin). These can be found in each cell.

The stainability of alpha-smooth muscle actin in myofibroblasts is temporary. Even in Dupuytren's disease the expression of alpha-smooth muscle actin seems to be intermittent. Myofibroblasts express alpha-smooth muscle actin in the proliferative

stage, and this attribute disappears in the residual stage. There is a significant correlation between the gradual contracture and the presence of alpha-smooth muscle actin-expressing myofibroblasts.

Myosin is the co-partner of actin in the connective tissue stress fibers (TOMASEK et al., 1999).

Integrin is a transmembraneous glycoprotein, which modulates cell to cell or cell to matrix contacts, therefore acting as a direct adhesive for molecules or as a receptor for protein ligands (MAGRO et al., 1997).

5.3 Glycosaminoglycans of the extracellular matrix

5.3.1 Structure and function of the glycosaminoglycans

To illustrate what the extracellular matrix looks like we will compare it with something quite different from digitopalmar fibrosis. The entire vitreous body of the eye with its apparently structureless, transparent gel consists almost exclusively of evolutionary young glucosaminoglycans with a distinct water-binding capacity and is therefore almost pure extracellular matrix. It becomes obvious from the vitreous body's gel like mass that glyocosaminoglycans contribute to the stability of the extracellular matrix. The physico-chemical stability (stiffness) of the extracellular space varies with its glycosaminoglycan composition.

Proteoglycans are extremely large molecules and make up the largest part of the extracellular ground substance.

Initially the term "proteoglycan" was reserved for molecules containing chondroitin sulfate firmly bound to non-collagenous protein. In the nineties, proteoglycans were redefined as a group of macromolecules typically consisting of a core protein to which at least one peripheral polysaccharide fragment, known as glycosaminoglycan chain, is covalently linked.

Small proteoglycan molecules have a rounded head filled with protein and a tail of glycan, which has awarded them the name "tadpoles" by SCOTT (1991). The "bottle brush-like" side chains of the large glycosaminoglycan macromolecules determined their biochemical and metabolic attributes as well as their name.

Glycosaminoglycans are the polysaccharide chains of the proteoglycans. Thus GAGs are polyanionic polymers of varying length consisting of repetitive series of disaccharide units. One component of these disaccharides is always an N-acetylated amino sugar (N-acetyl-glucosamine or N-acetyl-galactosamine), to which a sulphate group is bound. The second component is also a sugar, in general uronic acid.

The type, composition and GAG's profile are specific for the various types of connective tissue.

Proteoglycans used to be erroneously considered an independent biochemical class separate from glycosaminoglycans, but both terms are now used interchangeably. In the past, glycosaminoglycans were called mucopolysaccharides, a term no longer used.

Proteoglycan biosynthesis starts with the transcription and translation of the core protein in the endoplasmatic reticulum. The polypeptide and polysaccharide chains are merged in the Golgi complex. This is followed by the active extrusion of the large proteoglycan molecule into the extracellular matrix.

Glycosaminoglycans direct molecular interactions modulate the aggregation behavior, and influence viscosity and permeability. They are also responsible for the extracellular matrix's water-binding capacity and thus for possible swelling or turgor forces. The unique "entropy of dilution" is the ability of 1 g of glycosaminoglycan hyaluronic acid to bind 500 ml of water (VARMA et al., 1983).

In digitopalmar flexion contracture the water content increases progressively from apparently non-affected fascia to cords and finally to the nodule.

Water content in fresh normal fascia is 54.3%, in the seemingly unaffected palmar aponeurosis 59%, in the cord 60.7%, and in the glycosaminoglycan rich, Dupuytren nodule even 62.1% (BAZIN et al., 1980).

Biochemical attributes and structural and metabolic heterogeneity determine the distinction of glycosaminoglycans into four classes: (1) hyaluronic acid; (2) chondroitin and dermatan (and their sulfated derivates); (3) heparan sulfate and heparin; and (4) keratan sulfate (Figs. 11 and 12).

The heparin isomers and keratan sulfates are well known agents for anticoagulation, but it may be not that well known that they belong to the group of glycosaminoglycans. Only the first two classes of glycosaminoglycans are of importance for our further exploration of Dupuytren's disease.

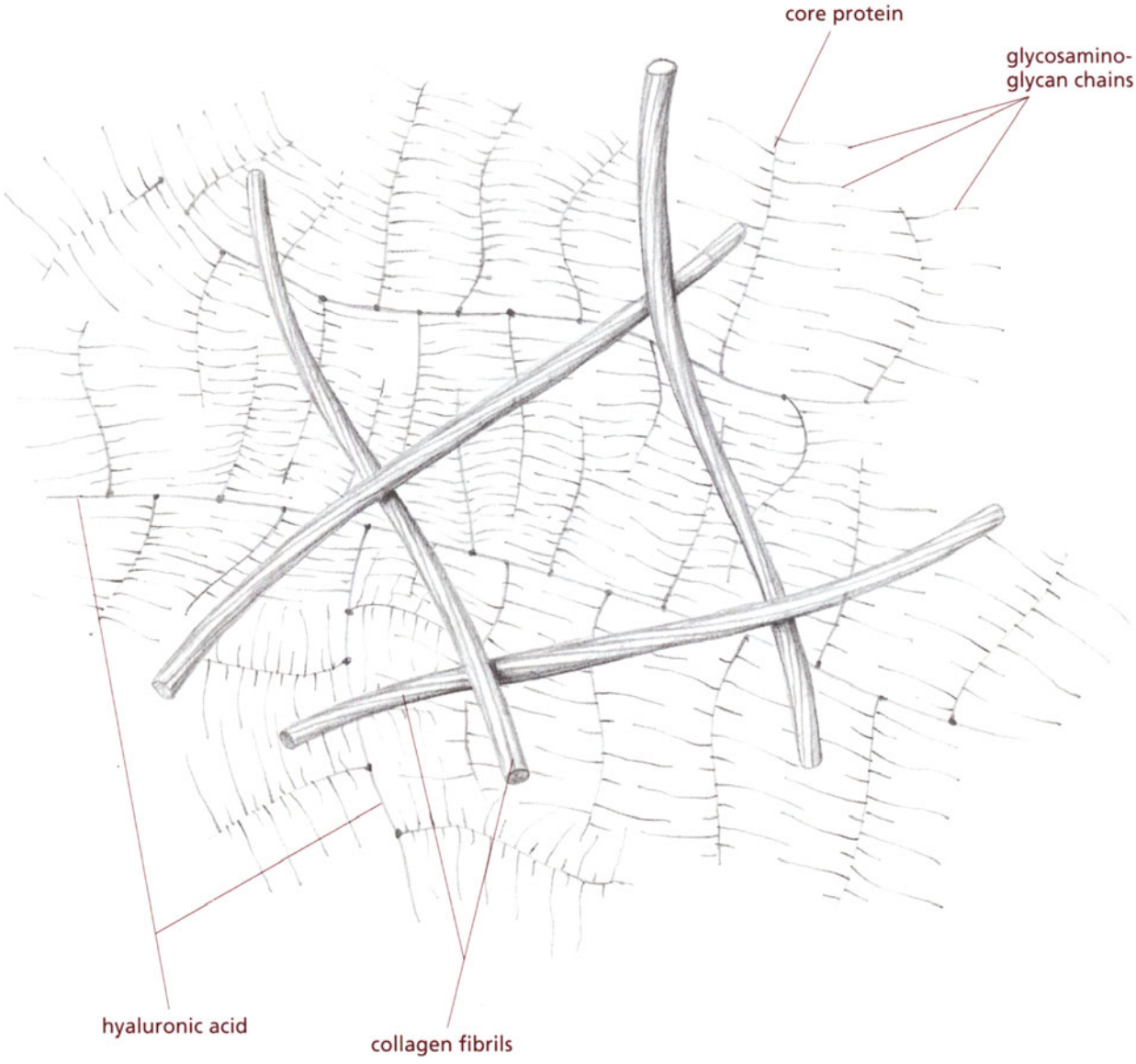


Fig. 11. Model of the extracellular space. The ground substance contains (nearly) no cells. The extracellular matrix is mostly composed of collagen fibrils and glycosaminoglycans. The glycosaminoglycan chains layered around the core protein form a "bottle brush" pattern.

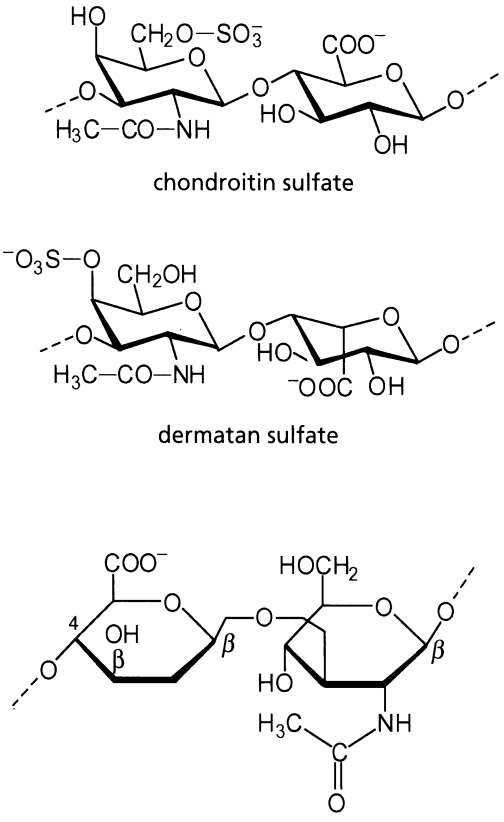


Fig. 12. Chondroitin sulfate, dermatan sulfate and hyaluronic acid are select glycosaminoglycans. Simplistically it may be said that glycosaminoglycans are long unbranched polysaccharide chains, composed of repeating disaccharide units, covalently linked to a central core protein. The peripheral polysaccharide fragment, known as glycosaminoglycan, provides the name for the entire complex. Because of its different structure, the evolutionary youngest hyaluronic acid is an "honorary" glycosaminoglycan.

5.3.2 Particularities of hyaluronic acid

The biochemical simplicity of hyaluronic acid identifies it as the earliest evolutionary form of glycosaminoglycan.

Hyaluronic acid is synthesized in many cell types, but primarily in the plasma membrane of fibroblasts.

Hyaluronic acid modulates the gel consistency of the extracellular substance and is responsible for its highly viscous plasticity (Bertheim et al., 1994). It also facilitates cell motility during tissue morphogenesis and participates in repairs. Hyaluronic acid is an indicator for the repetitive remodeling of the connective tissue.

When the restructuring process is finished, surplus hyaluronic acid is degraded by the body's own enzyme hyaluronidase. The degradation products of hyaluronic acid stimulate angiogenesis (Bloom et al., 1986; Mast et al., 1992; Brenner, 1994).

Despite a serum half-life of less than five minutes hyaluronic acid can be found in blood serum. Its estimated daily turnover is 150 mg. The concentration in urine is one tenth of the serum concentration, and most of the remainder is excreted extrarenally. Hyaluronic acid is primarily cleared by the liver (Endo et al., 1979; Respondek et al., 1989).

High plasma concentrations of hyaluronic acid are found in patients with rheumatoid arthritis, especially early in the day during the phase of morning stiffness (Trelstad, 1992).

5.3.3 Chondroitin and its analogues

Chondroitin and its derivatives are the most important and best investigated glycosaminoglycan components (Gurr et al., 1985; Karlson, 1988).

Dependent on the presence and position of the sulfate groups, three chondroitin isomers are distinguished: non-sulfated chondroitin, chondroitin-4-sulfate, and chondroitin-6-sulfate.

The chemical characteristics of chondroitin sulfate and dermatan sulfate are similar. The main difference is the epimerisation from glucurone into irudonic acid.

The metabolism of all glycosaminoglycans is high. The biological half-life of chondroitin sulfate is seven to 10 days (Trelstad, 1989; Buddecke, 1989).

5.3.4 The dermatan group

With respect to Dupuytren's disease we are interested in these dermatans: non-sulfated dermatan, dermatan-4-sulfate, and dermatan-6-sulfate.

High concentrations of dermatan are found in human skin and in other locations. Dermatan sulfate is a naturally occurring anticoagulant glycosaminoglycan, which is a potential therapeutic alternative to heparin (Dawes, 1992; Silvestro, 1992).

Varma et al. (1983) state that dermatan sulfate is 10 Ångström (1 nanometer) long.

The nomenclature of dermatan and dermatan sulfate is misleading as both are occasionally re-

garded as mere derivates or modifications of the previously mentioned chondroitin group.

5.4 Fascial and plasmatic patterns of glycosaminoglycans in Dupuytren's disease

Tissue affected by digitopalmar contracture contains proteoglycans of the small (PG-S) and large type (PG-L), and also free glycosaminoglycan chains from the chondroitin group and dermatan sulfates. The proteoglycan ratio is about 85:15% (PG-S/PG-L). Structural analyses imply that in Dupuytren's disease, PG-L has a longer core protein and a lower concentration of glycosaminoglycan. In comparison with the healthy fascia, the diseased fascial tissue appears to be not only characterized by a quantitative increase in glycosaminoglycans or proteoglycans, but also by their structural changes. Whether the core protein or the synthesizing cells control the structure of glycosaminoglycan has not been resolved (Schmidtchen et al., 1992; Gurr et al., 1994; Scott, 1994).

If the palmar aponeurosis is examined by proximal, distal, radial, and ulnar segments, significant differences in the content of hyaluronic acid become obvious, in proximal-distal direction ($p < 0.05$) and between the ulnar and distal fascia ($p < 0.05$). The chondroitin content shows a significant increase from proximal to distal ($p < 0.05$), and chondroitin-4 sulfate increases from proximal to radial ($p < 0.05$) and from proximal to distal ($p < 0.05$) but also from radial to ulnar. Significant concentration differences also exist for dermatan-4-sulfate from proximal to radial ($p < 0.05$), from proximal to ulnar ($p < 0.05$), and from proximal to distal (Fig. 13a).

Nine significant variables therefore point to similar glycosaminoglycan concentration gradients in distal and ulnar and radial direction (5x distal, 2x radial, 2x ulnar (Fig. 13b).

These findings correspond to a detailed description of repetitive repair processes in the extracellular matrix. The biosynthesis of glycosaminoglycan starts with hyaluronic acid, proceeds via chondroitin, and results in chondroitin-4 sulfate after sulfatation, which is then turned into chondroitin-6-sulfate and its epimer. This explains the high proportion of hyaluronic acid of 41.5–63.7%. The fascial glycosaminoglycan with the second highest concentration is dermatan-4-sulfate. Its relative concentration is 17.9–24.1%. Dermatan-4-sulfate correlates with the clinical stage of the ulnar types of Dupuytren's disease. The total GAG content of digitopalmar fascia with Dupuytren's disease is highest both in the ulnar and distal segments.

In the radial types, the maximum total concentration of glycosaminoglycan is also found distally, followed by, in decreasing order, the ulnar, radial, and proximal location. Hyaluronic acid was most excessively increased, with a maximum of 66.3 mmol/g in the ulnar location and the lowest value distally.

These results illustrate how repetitive fascial remodeling and maintenance processes cause a release of glycosaminoglycans from the extracellular matrix into the lymphatic or blood system. The fragments are most commonly broken down in the endothelial cells of the liver, where they are bound to receptors, endocyted and lysomally digested (Janzen, 1992).

Plasma hyaluronic acid levels are also increased to more than eight times the normal value. This is especially remarkable, as hyaluronic acid is known to have a very short half-life.

If one compares hyaluronic acid plasma concentrations to concentrations of chondroitin isomers, however, it becomes obvious that the excess chondroitin and the scarce chondroitin-6-sulfate make up the majority of glycosaminoglycan components in plasma, i.e. 93.3%. The ratio of chondroitin to chondroitin-6-sulfate is 1:6.37.

These proportions mirror the biosynthesis. The high concentrations of chondroitin isomers can be explained with the fact that terminal sulfatation is unusual and nearly non-existent in blood. Sulfatases are usually found inside of cells or cell bound in fascial tissue. This is also the main reason why dermatan-4-sulfate and dermatan-6-sulfate are not detectable in serum (Fig. 14).

If one limits the possible conclusions to be derived from serum concentrations to just Dupuytren's disease, disregarding if the patient has the ulnar or radial type, then eight discriminating serum variables—namely, hyaluronic acid, dermatan-4-sulfate, chondroitin, uronic acid, chondroitin-6-sulfate, dermatan-6-sulfate, and dermatan—suffice to diagnose stage IV disease with a 91.7% probability.

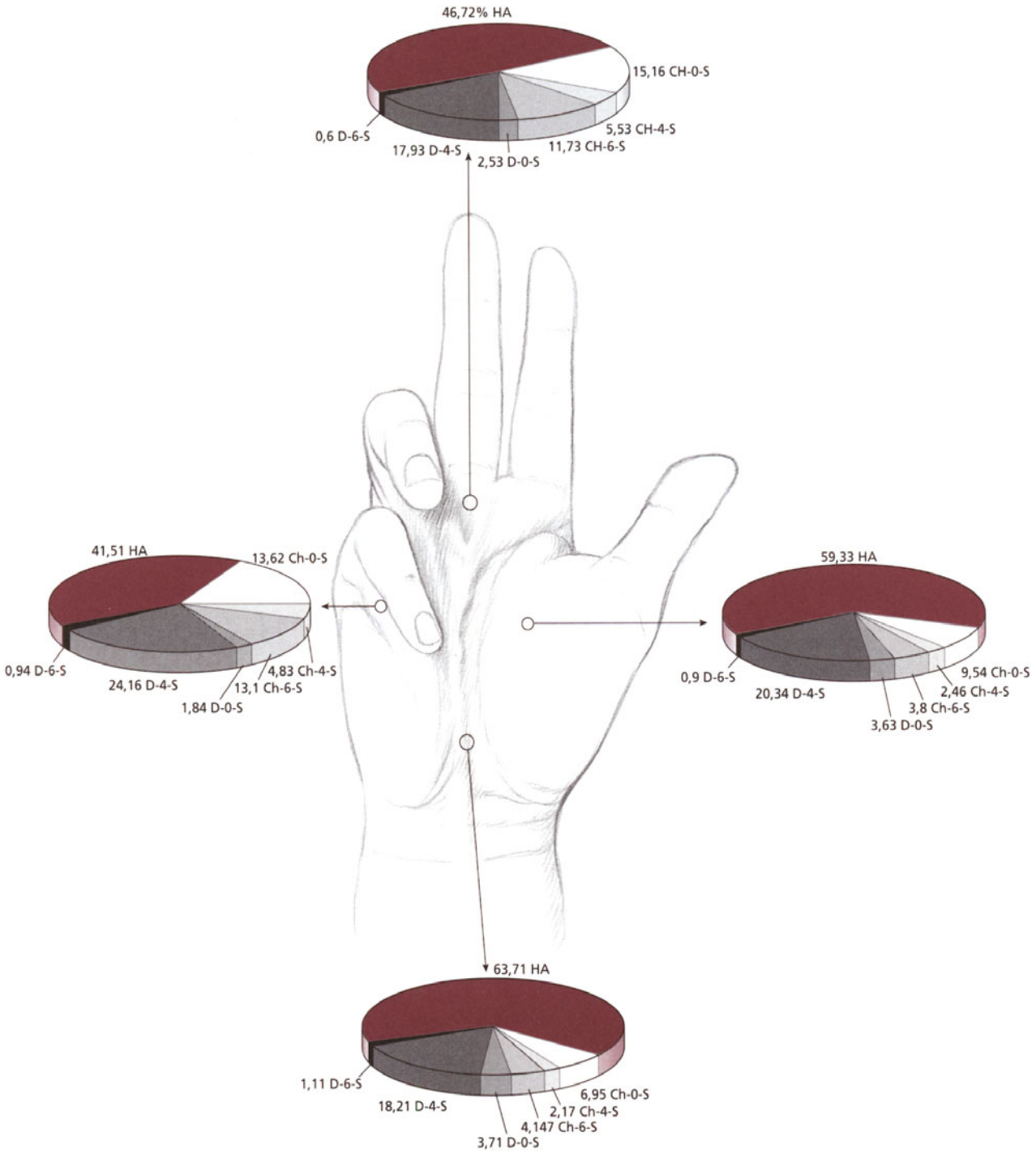


Fig. 13a. Parallel to the mechanical strain and the profile of the digitofascial cord, glycosaminoglycan concentrations showed the following gradient: hyaluronic acid decreases significantly from proximal to distal ($p < 0.05$). Chondroitin, however, increases in the same direction ($p<0.05$), and chondroitin-4-sulfate increases notably from proximal to radial ($p < 0.05$), proximal to distal ($p < 0.05$) and radial to ulnar. Significant concentration differences of dermatan-4-sulfate are also seen: in a proximal-radial direction ($p < 0.05$), from proximal to ulnar ($p < 0.05$), and from proximal to distal ($p < 0.05$). Nine significant variables indicate a glycosaminoglycan concentration gradient in a distal, ulnar and radial direction.

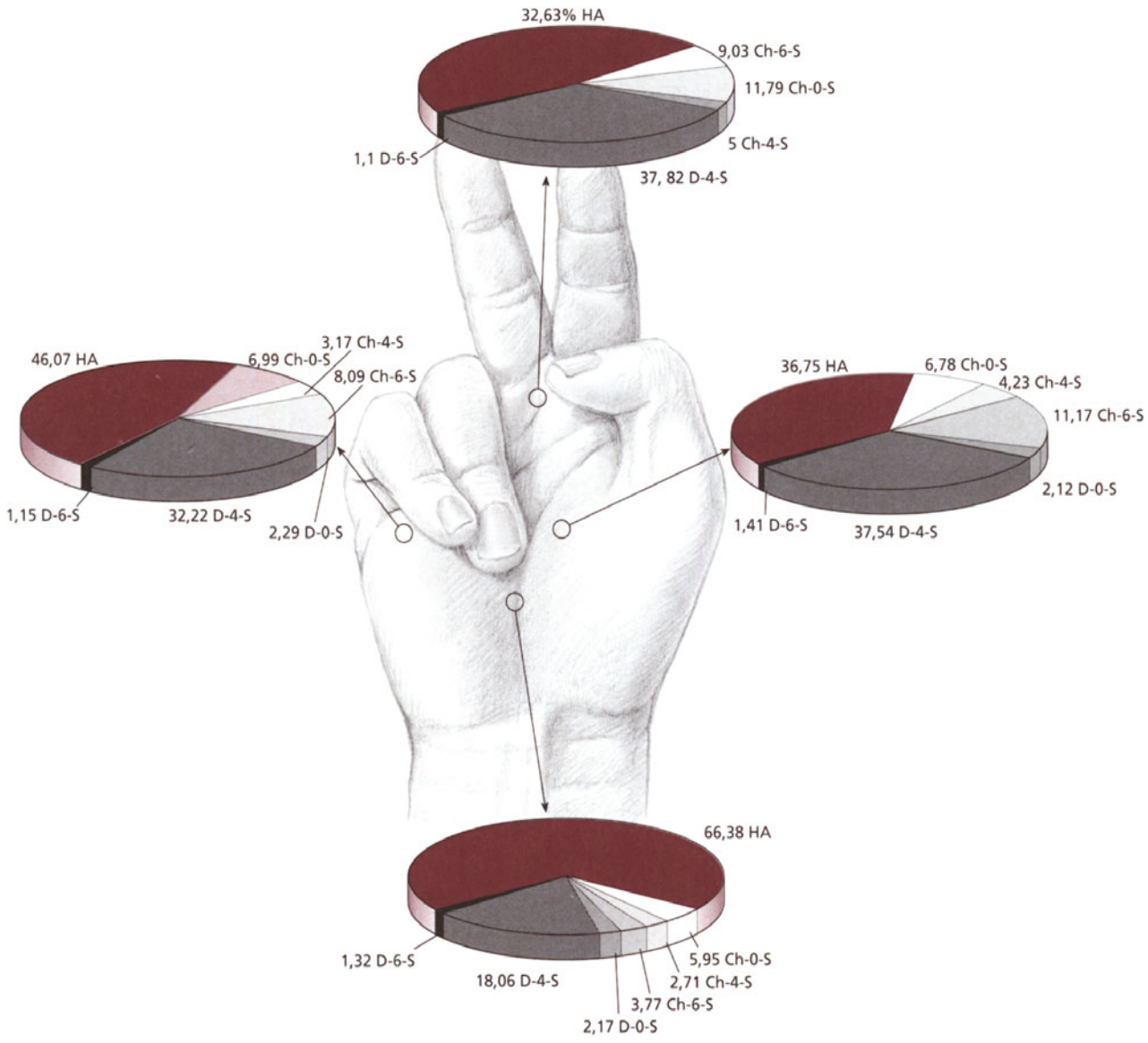


Fig. 13b. The radial type of Dupuytren's disease therefore appears to be a more expansive (widespread) ulnar type.

It has to be stressed once again that a definite diagnosis of Dupuytren's disease can be made from only eight glycosaminoglycan plasma concentrations without knowing the clinical status and without having seen the patient.

For the first time this allows to diagnose the systemic connective tissue disorder Dupuytren's disease reliably, as it can be done with other connective tissue disorders, such as rheumatism.

The histologically identical ectopic connective tissue deposits in the planta pedis, penis, and Garrod's knuckle pads can also be adequately explained on the basis of the plasma results.

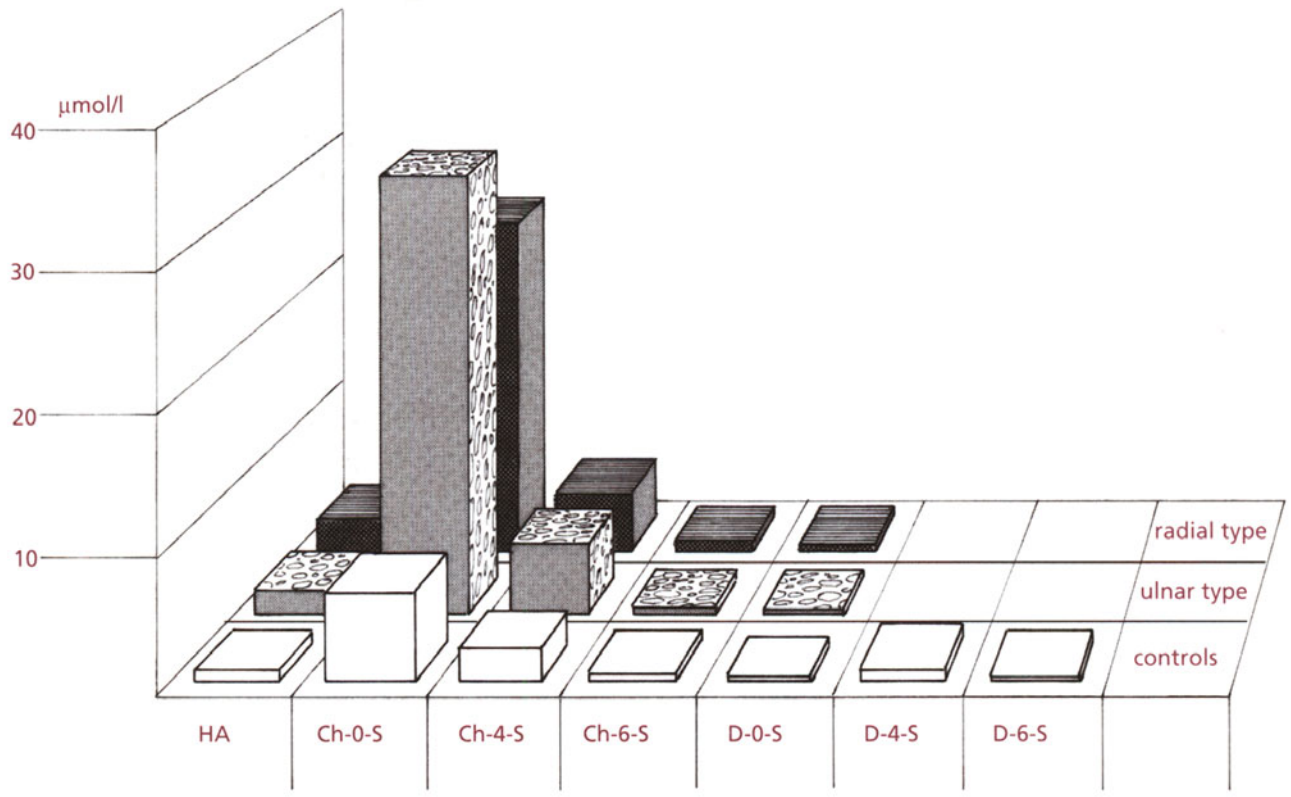


Fig. 14. Plasma hyaluronic acid concentrations are eight times higher than normal. This corresponds to an ongoing remodeling process. If plasma levels of hyaluronic acid are compared with those of chondroitin isomers, it becomes obvious that nearly no chondroitin-6-sulfate, but chondroitin is the predominant glycosaminoglycan in plasma; it presents 93.3% of the plasma glycosaminoglycans. The ratio of chondroitin to chondroitin-6-sulfate is 1/6.37. The high concentration of chondroitin isomers is explained by the fact that no final stage sulfatation occurs in blood, because there are only intracellular sulfatases and those cell-bound in the fascial tissue. This is also why no dermatan-4-sulfate and dermatan-6-sulfate are found in the plasma of patients with the radial and ulnar type. If only blood samples are used, eight discriminatory variables are sufficient to determine with a 91.7% probability if an individual has Dupuytren's disease stage IV or not (discriminant analysis). These discriminatory variables are as follows: hyaluronic acid, chondroitin-4-sulfate, chondroitin, uronic acid, chondroitin-6-sulfate, dermatan-6-sulfate, dermatan-4-sulfate and dermatan.

5.5 Interactions between collagens, glycosaminoglycans, and fibroblasts

On the basis of the results presented, the contraction mechanism between fibroblasts, their many variants, collagen fibers of the extracellular ground substance, and glycosaminoglycans can be interpreted as follows:

Ryan et al. (1974) assumed that an endogenous mediator causes the myofibroblast to contract. The "lock step" system provides the static end point for the joint contraction of all myofibroblasts. This means that changes in the three-dimensional makeup of collagen commence during the contraction, which translates into extracellular collagen "shortening." To safeguard the contraction result, gradual biochemical restructuring occurs.

Glycoproteins are the cement substance and hold the collagen fibrils together (Gabbiani et al., 1977).

Fibrillogenesis and collagen fibril growth can be associated with specific glycosaminoglycans:

small diameter fibrils coincide with a high concentration of hyaluronic acid, medium size fibrils coincide with a high concentration of chondroitin, and large-caliber collagen fibrils coincide with the highest concentration of dermatan sulfate (PARRY et al., 1982).

There is experimental proof that inside the fibrils, proteoglycans (= glycosaminoglycans) are linked to collagen. Each proteoglycan has several, transfibrillar loci, comparable with docking sites. The selectivity between the docking site and a special fibrillar type is high. If there is no match, the docking site remains unused (SCOTT, 1986; 1994).

TOMASEK (1986, 1987) proved that the myofibroblasts arising from Dupuytren tissue use a non-muscle myosin to start contraction processes, instead of the expected smooth muscle myosin.

Extracellular factors, such as transforming growth factor (TGF) β, glycosaminoglycan heparin and gamma-interferon, cause the dispersion of alpha-smooth muscle actin. Actin is one of the main components of the long, intracellular microfilaments that stimulate myofibroblasts to contract (TOMASEK et al., 1995).

6 Subjective complaints

Precursors of the actual onset of Dupuytren's disease are a burning sensation, itching, and tension (Hauer et al., 1978; Meyer, 1991). Increasingly, patients complain about localized nerve irritations, impaired fitness for use, or the inability to work as a result of the deforming flexion contracture (Buck-Gramcko, 1976; Flint, 1990).

The contracted fingers cause problems when donning gloves, putting hands in pockets, and hinder social contacts such as shaking or squeezing hands (Haimovici, 1978).

Patients report that they poke themselves in the eye with the contracted fingers when washing their face or shaving (Krause-Bergmann, 2000).

7 Symptoms of Dupuytren's contracture

Funnel shaped disturbances of the skin relief (skin pits) in the vicinity of the proximal or distal creases characterize the early stage of the disease (MAILÄNDER, BRENNER et al., 1994).

These U-shaped compression zones are distally open. JOHNSON (1980) named them eponymically and confidently the Hugh Johnson sign (Fig. 15).

Occasionally, and incorrectly, patients begin to pay particular attention to their palm after a minor trauma, and associated this with the disease onset.

Digitopalmar nodules or moderate cords are initially palpable, which may sometimes already limit finger extension (MEISTER et al., 1978) (Fig. 16a).

Subsequently visible or palpable longitudinal cords with areas of induration develop (BUCK-GRAMKO et al., 1992) (Fig. 16b–16e). Cord formation of the natatory ligament leads to contractions in the interdigital spaces, so that the primarily U-shaped web spaces distort into a V shape and result in a limited hand span.

In the late stage the "prayer sign" occurs (CRIPS, 1991; ROSENBLOOM et al., 1996).

Classically, ring, middle and little finger are affected with decreasing frequency (BRENNER, 2001). This represents the ulnar type of Dupuytren's disease.

When the thumb and index finger are affected, the disease takes a different course, has different recurrence rates, and different functional results after surgery. Therefore, MEYERDING (1936), TUBIANA and DEFRENNE (1976), NETZELOF (1986), TUBIANA (1990), and BRENNER (1994) refer to this as the radial type of Dupuytren's disease.

Data about the incidence of the radial type vary between 3.3% and 11% (JAMES et al., 1952: 3.3–5%; MILLESI, 1959: 8.3%; MACCULLUM et al., 1962: 10%; NISSENBAUM et al., 1980: 11%). In contrast, KALBERG (1935/36) and TUBIANA and DEFRENNE reported a 32–39% incidence. They believe that the process was previously underreported because subtle findings had been overlooked.

As a result of our own studies on fascial and plasmatic glycosaminoglycan components, we conclude that the ulnar and radial types can be differentiated clinically, but the radial type is not an independent entity, it is a more expansive form of the ulnar type.

Fig. 15. The chronology of Dupuytren's disease. Onset: distally open and U-shaped disturbance of the skin relief (Hugh Johnson sign) over the distal palmar crease of the ring finger.

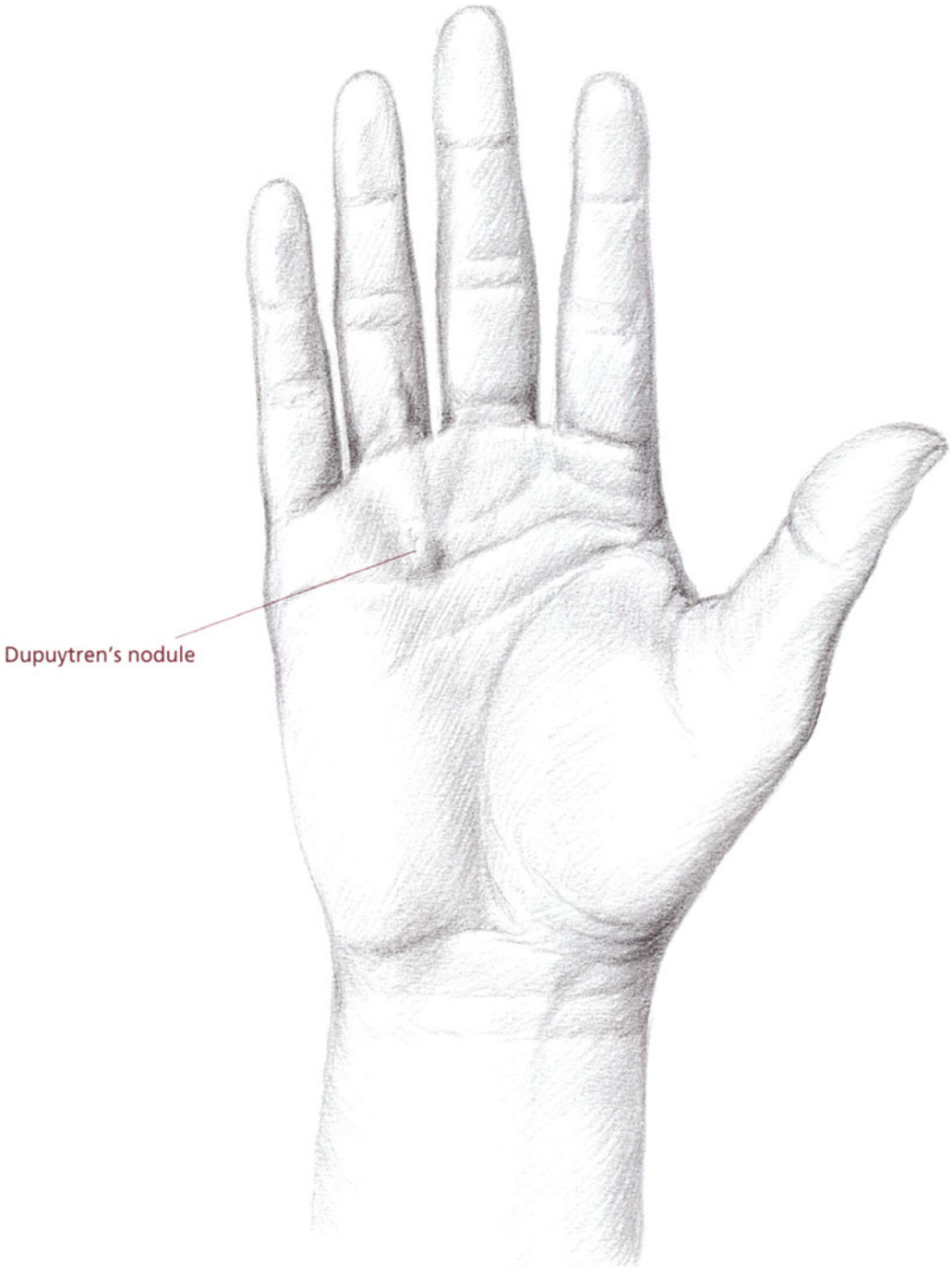


Fig. 16a. Nodular stage of digit IV and its corresponding palmar segment with unrestricted extension (stage N).

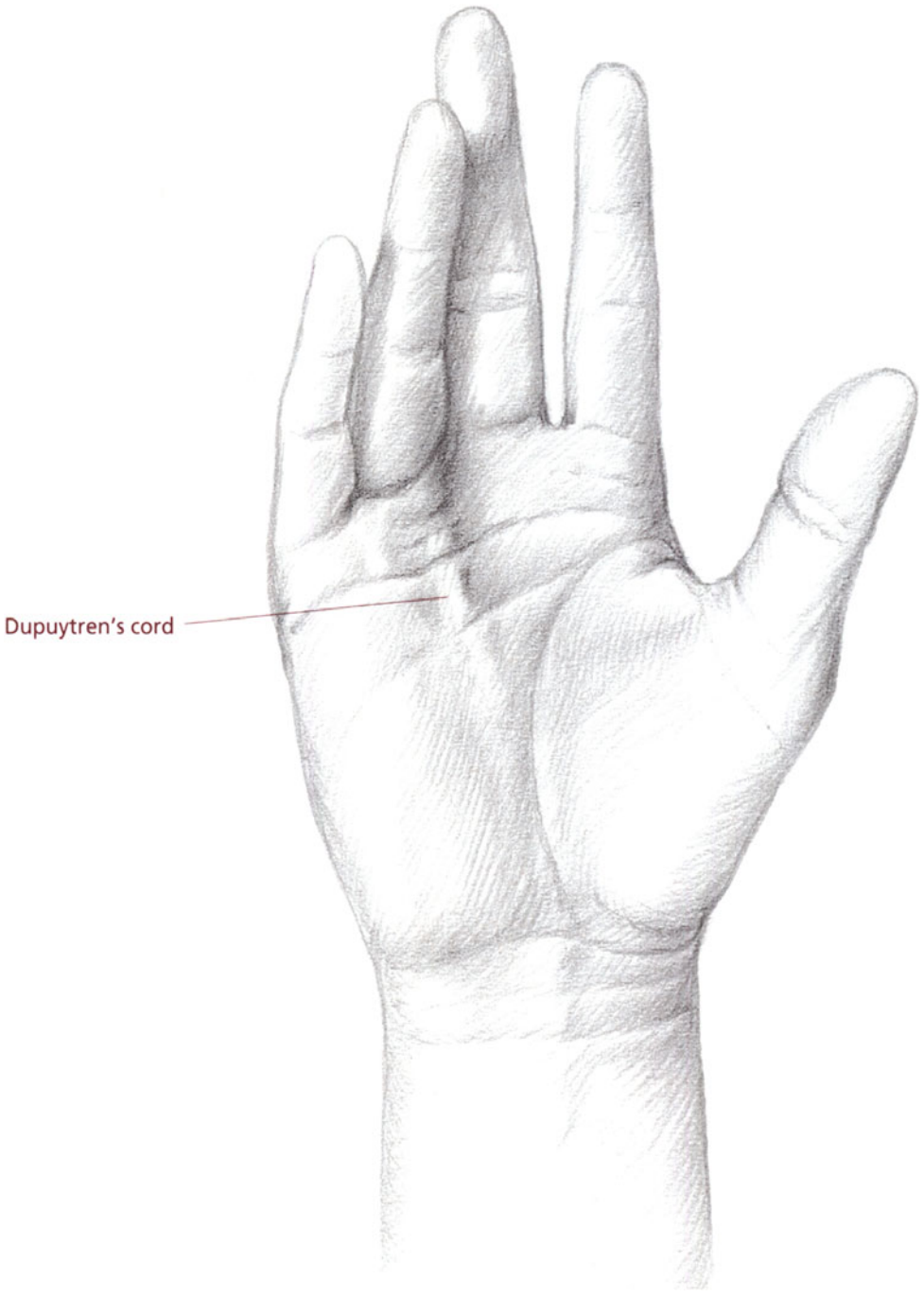


Fig. 16b. Initial cord stage with puckered palmar skin and beginning flexion contracture (stage I).

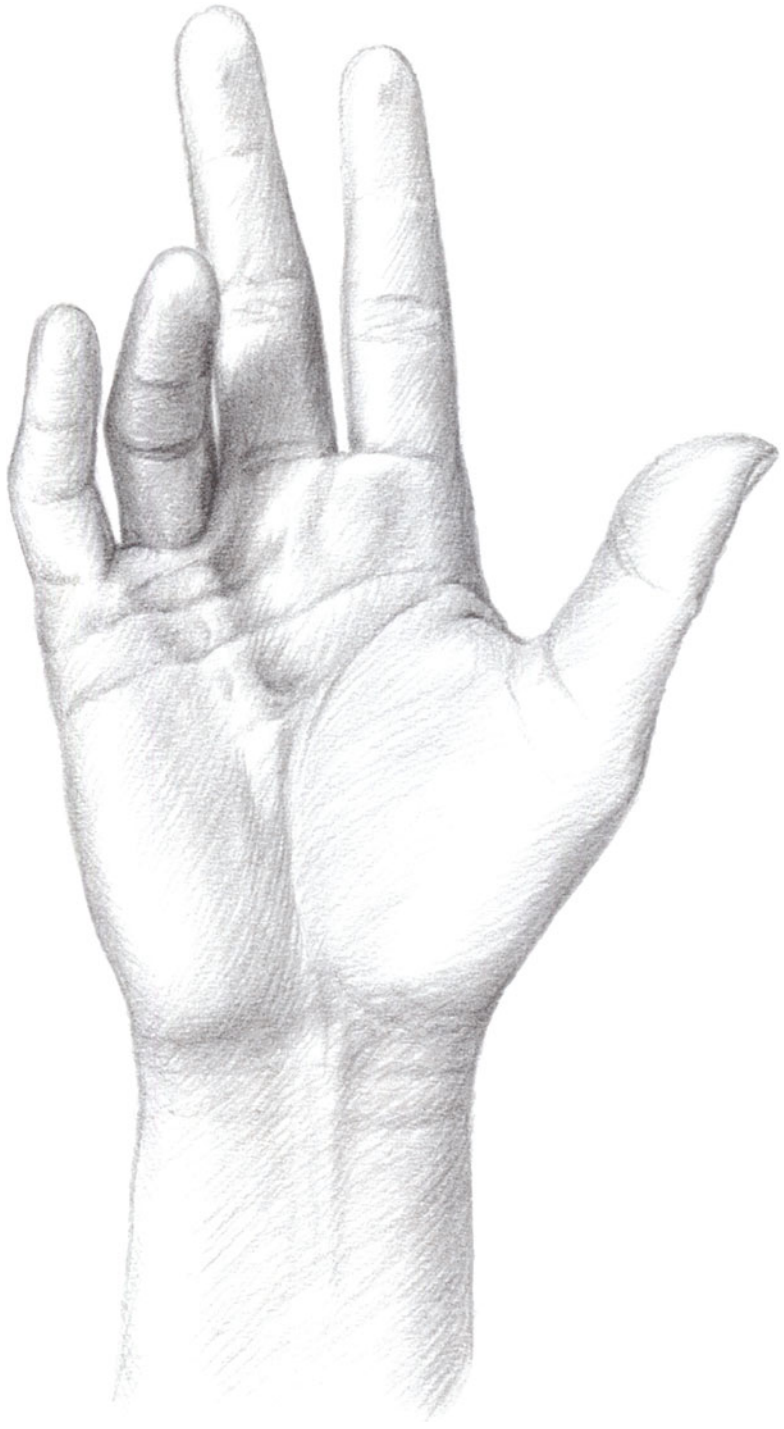


Fig. 16c. Cord formation with flexion contracture of ring finger; also beginning on the little finger. The lack of extension is less than 90 degrees (stage II).

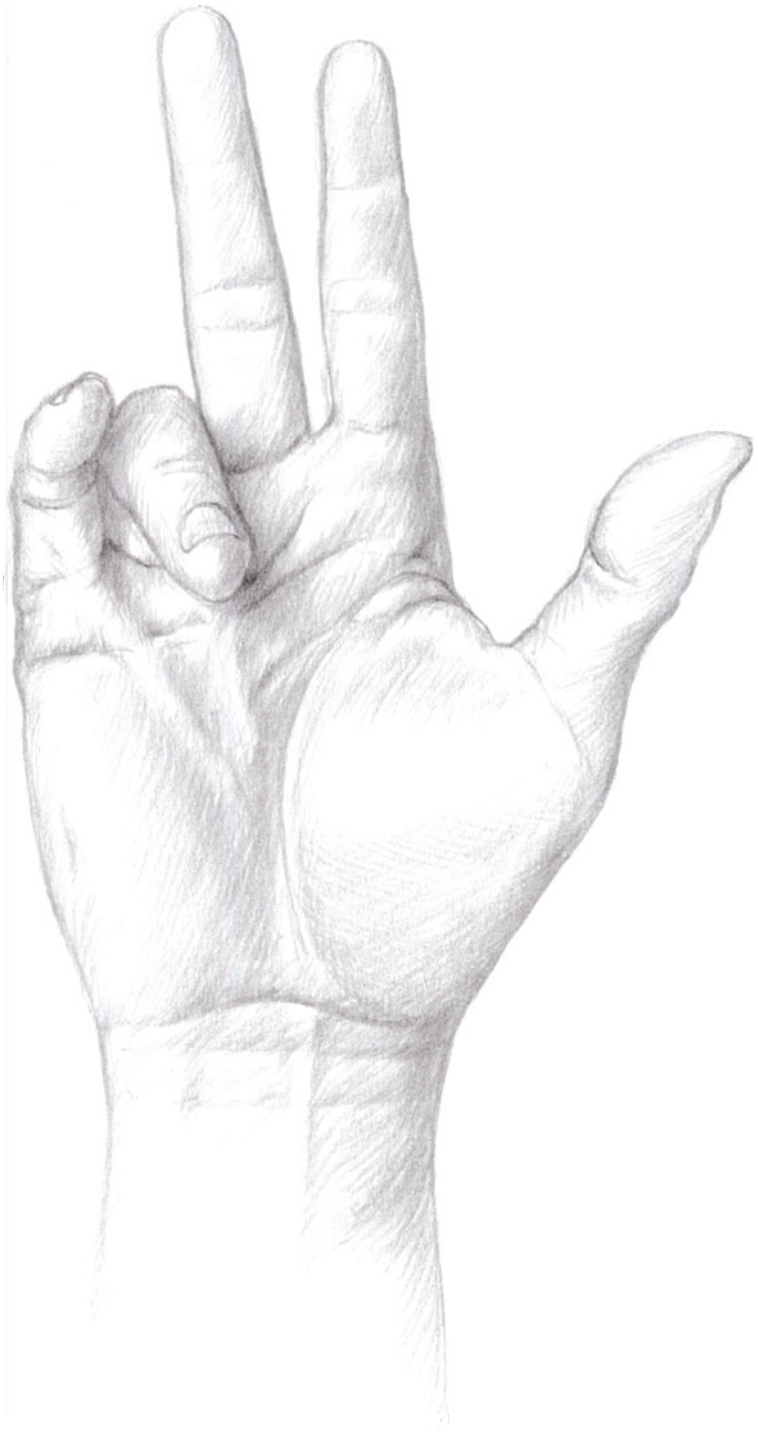


Fig. 16d. Flexion contracture between 91 and 135 degrees measured as the total sum of all extension deficits among the fingers affected by Dupuytren's disease (stage III).

Fig. 16e. Contracture of the ring and little finger with both PIP4 and PIP5 joints fixed and hyperextension of DIP5. Moderate extension deficit of the middle finger (stage IV).

8 Course of Dupuytren's contracture

Dupuytren's disease can either progress slowly, being interrupted by phases of stagnation, or it can cause contracture-induced dislocations of the digital joints within a short time (MILLESI, 1959, 1965; MIKKELSEN, 1977). This is dependent on a person's individual disposition (STRICKLAND et al., 1990).

In addition to usual disease stages, special subtypes need to be mentioned because of the course they follow: (1) the particularly "mild" variant in patients with diabetes mellitus; (2) an aggressive form of Dupuytren's disease, which is mainly observed in young people (30–40 years of age) and takes an expansive bilateral course (radial and ulnar) (HUESTON, 1982; BELUSA et al., 1988; LAMB, 1981; MCFARLANE, 1990).

CHIU et al. (1978) also distinguish between early stage disease, the active stage, and advanced Dupuytren's disease. The first form is characterized by nodule formation without finger retraction. When the flexion contracture increases the active stage has been reached, and an increasing retraction corresponds to advanced Dupuytren's disease.

On the basis of the clinical courses analyzed by MILLESI (1959), the digitopalmar flexion stage is not necessarily inevitable. In recognition of these findings and in order not to prejudice, MACCULLUM et al. (1962) suggested naming the disease not Dupuytren's contracture, but instead, as in the French "maladie de Dupuytren", Dupuytren's disease.

9 Anatomy of the palmar fascia (G. M. Rayan)

Familiarity with the subtle constituents of the complex palmar fascial anatomy of the hand is necessary to understand the convoluted pathologic changes that take place in Dupuytren's disease and the transformation from a normal to a pathologic anatomy.

The following chapter encompasses information about the embryologic development, gross and fine anatomy of the palmar fascial complex, and its relationship to Dupuytren's disease.

9.1 Development of the palmar fascia

Development and differentiation of the hand takes place between 4 and 8 weeks following gestation. The mesenchymal cells of the limb bud lateral plate mesoderm give rise to fascial structures, connective tissue, tendons, ligaments, bones and vessels of the upper limb. The palmar fascia develops from the superficial mesenchymal layer adjacent to the palmarly located AER (= Apical Ectodermal Ridge). Stack (1973, 1985) examined serial sections of the embryonic palmar fascia between 16 and 18 weeks of fetal life. He observed condensed bundles of longitudinal fibers within the palmar aponeurosis, which are the precursors of the pretendinous bands. Caughell, McFarlane, McGrouther et al. (1988), conducted a study of the embryonic palmar fascia from the fifth gestational week to term using trichrome staining and light and electron microscopy. They identified, at the fifth week, transverse and longitudinal fibers of a predominantly cellular palmar aponeurosis along with septa of Legueu and Juvara, which arose from the transverse fibers. At the twelfth week the pretendinous bands became apparent. When present, the palmaris longus tendon was in direct continuity with the longitudinal fibers of the palmar aponeurosis, with a transition from tendon to fascia over the transverse carpal ligament. Other fascial structures such as natatory ligament, Cleland's and Grayson's ligaments were not clearly defined.

Two opposing views exist regarding the phylogenetic status of the palmar aponeurosis. The first school of thought advocates that the fascia is homologous to the palmaris longus (Wood Jones, 1941; Le Gros Clark, 1958) and the second advocates that both structures have different origins and their connection is incidental (Kaplan, 1966; Caughell, 1988). Wood Jones (1941) hypothesized that the palmaris longus muscle fibers used to be a superficial digital flexor muscle, which had tendons that extended to the hand and bifurcated in order to flex the metacarpophalangeal joints. Le Gros Clark (1958) also hypothesized that the palmaris longus muscle was a digital flexor in mammals and its distal part degenerated into the palmar aponeurosis. The palmar aponeurosis, with its five strips—it now has supportive function to the palm—may represent remnants of degenerated palmaris longus tendons. For the Camitz transfer, the palmaris longus tendon can be elongated by extending the dissection into the palmar aponeurosis. Lewis (1901) suggested that embryologically, the forearm flexor muscles develop as two separate groups, superficial and deep. The superficial muscle group consists of the flexor carpi radialis, pronator teres and palmaris longus. The deep group consists of the flexor carpi ulnaris, flexor digitorum superficialis and flexor digitorum profundus muscles. He hypothesized that the palmaris longus was most likely a wrist rather than finger flexor.

Caughell et al. (1988) suggested that the palmar aponeurosis was a fascial structure to begin with and not a tendinous extension of the palmaris longus. They found no phylogenetic relationship between the palmaris longus and palmar aponeurosis. They considered them to be separate

anatomic entities as demonstrated by the different staining properties of the tendon and palmar aponeurosis. KAPLAN (1966) also believed that the connection between the palmaris longus and palmar aponeurosis is incidental by anatomical location. According to the second school of thought, the palmar aponeurosis is probably not disappearing because it is not without function. The palmar aponeurosis retains the flexor tendons and this retinacular function stabilizes the metacarpals and support the palmar skin against compressive and shear forces. The palmar fascial system of the hand and digits provides anchorage for the mobile palmar skin in different postures of the hand. RITTER (1973) suggested that the palmar fascia has a protective function to the deeper structures and maintains the transverse arch and helps in cupping of the hand by its fixation to the metacarpals. MANSKE and LESKER (1983) suggested that the palmar aponeurosis transverse fibers proximal to the A_1 pulley have a retinacular function by forming a pulley structure that retains the flexor tendons.

Some authors believe that there is a relationship between palmaris longus and Dupuytren's disease. It was suggested that Dupuytren's disease rarely develops in the absence of the palmaris longus (NIEMINENS, 1986), but there is also a very significant chance of developing Dupuytren's disease if the palmaris longus is present (POWELL et al., 1986). Tension transferred from the palmaris longus to the palmar aponeurosis may be a causative factor. Dupuytren's disease, however, can occur in the absence of the palmaris longus. FAHRER (1980) noted a connection between the flexor carpi ulnaris and palmaris longus. The terminal fibers from the flexor carpi ulnaris join the palmaris longus, and in the absence of the palmaris longus the flexor carpi ulnaris completely takes over the longitudinal tension of the palmar aponeurosis. Assuming that this hypothesis is true, then it is likely that the flexor carpi ulnaris is the precursor of the palmar aponeurosis, rather than the palmaris longus; and the palmar aponeurosis' function has adapted to become retinacular in nature. The ulnar side of the hand is more often involved in Dupuytren's disease than the radial side and Dupuytren's disease may extend to the flexor carpi ulnaris tendon. During power grasp, the wrist has a natural tendency to deviate ulnarly so that the flexor carpi ulnaris tendon is aligned with the longitudinal pretendinous band fibers of the ulnar digits. I have seen one patient with severe typical Dupuytren's disease in one hand and nearly absent contralateral disease. The contralateral extremity was affected by an untreated chronic high ulnar nerve injury with paralysis of the flexor carpi ulnaris and intrinsic muscles. Congenital absence of palmar aponeurosis without ectrodactyly has not been reported, but congenital abnormalities of the palmar aponeurosis may be present in conditions associated with anomalous creases, such as chromosomal abnormalities.

9.2 Anatomy of the palmar fascia

The anatomy of the palmar facial structures of the hand can be studied at three levels, gross, fine and microscopic. The fine anatomy is studied by using a magnification greater than loupe magnification. An operative microscope would be necessary to visualize detailed structures that are difficult to see by the naked eye or with loops. An arthroscope can also enhance the ability to visualize deep and distant anatomic structures without disturbing the integrity of the fascial structures.

The fine palmar fascial anatomy is most appropriately studied using fresh frozen human cadaveric hands. I studied the fine palmar facial anatomy using an operative microscope (Carl Zeiss, Inc., Oberkochen, Germany) and a small arthroscope (Dyonics, Andover, MA). The arthroscope allowed examination of the partially reflected palmar aponeurosis and the undisturbed septa of LEGUEU and JUVARA before and after removal of the flexor tendons (Fig. 17). The fascial anatomy of the palmar aspect of the hand and digits, hereafter referred to as the palmar fascial complex, was found to be a composite system that can be divided into five anatomical components. This classification is the consequence of fine anatomical dissections, literature reviews and surgical observations by the author (RAYAN, 1999). Excluding the transverse carpal ligament, this palmar fascial complex (PFC) consists of the radial (thenar) aponeurosis, ulnar (hypothenar) aponeurosis, central (palmar) aponeurosis, palmodigital (synonymous: digitopalmar) fascia and digital fascia (Fig. 18a, b). The term palmar fascia is often used interchangeably with palmar aponeurosis. A distinction, however, is made here between the palmar aponeurosis and the palmar fascia which encompasses the three

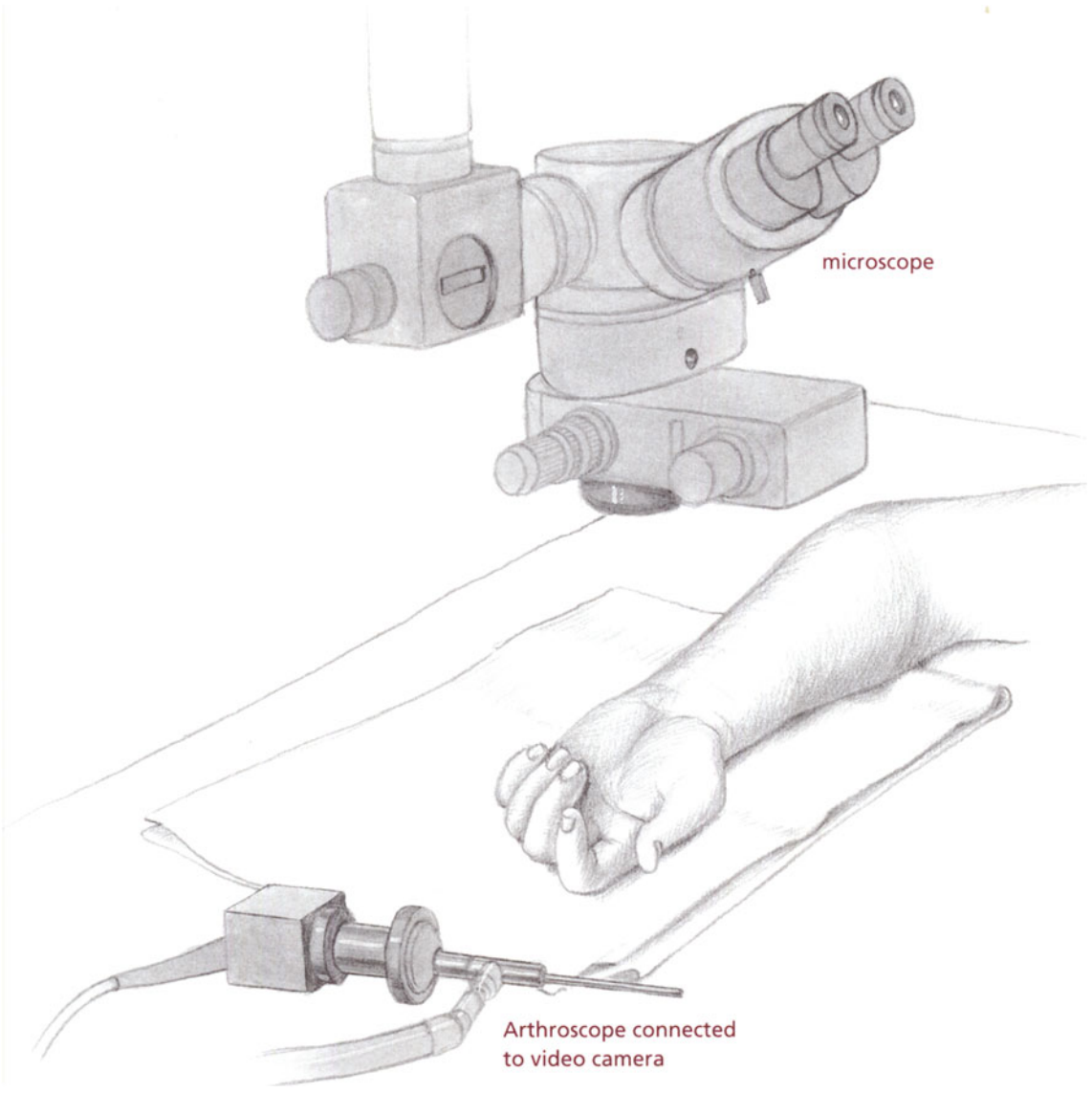


Fig. 17. Operating microscope and a small arthroscope are valuable tools to visualize microdissections of the palmar fascial complex. This technique permits the detailed examination of the intact vertical septa of Legueu and Juvara as well as other fascial structures.

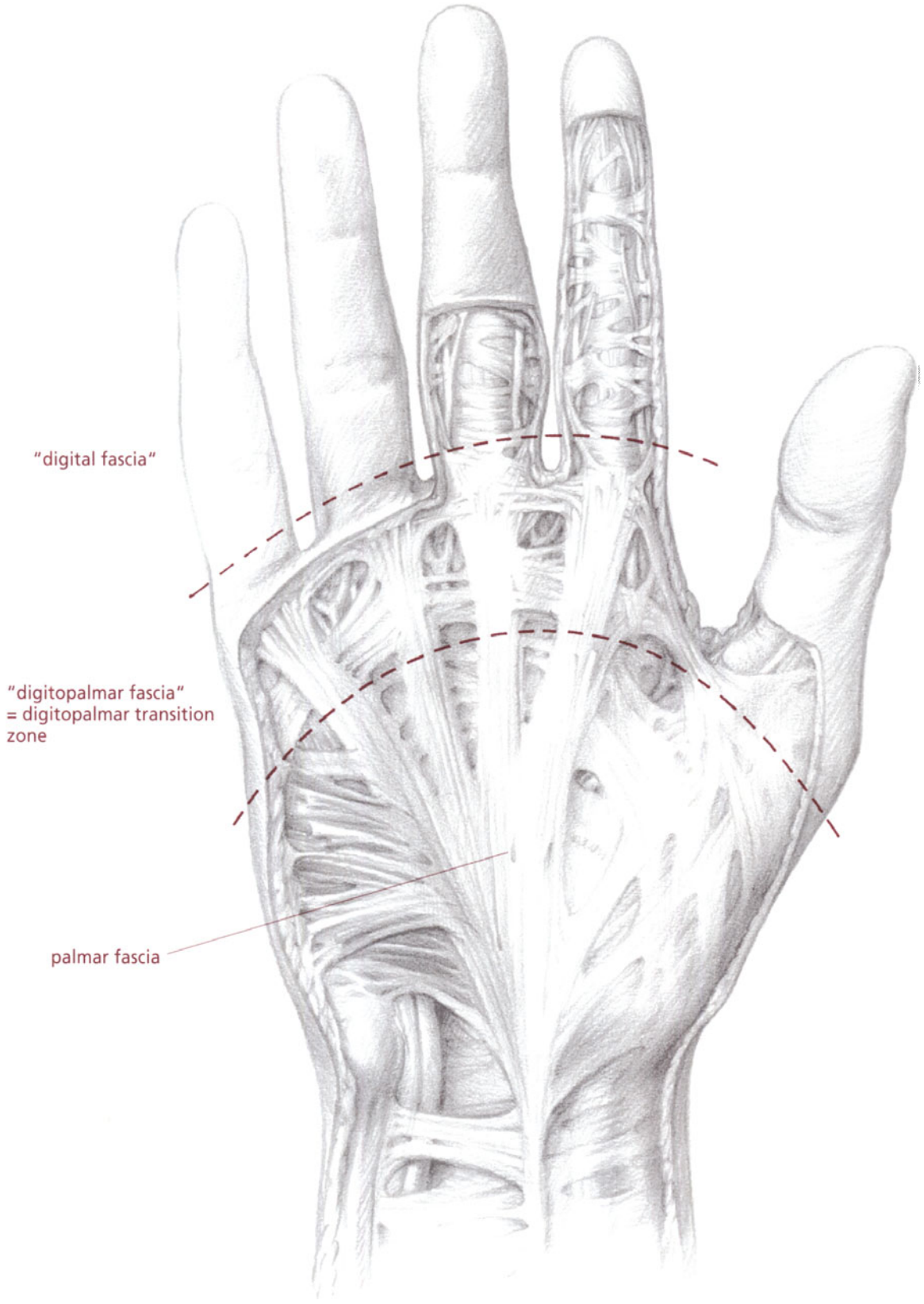


Fig. 18a. Zones of the palmar fascial complex (PFC)

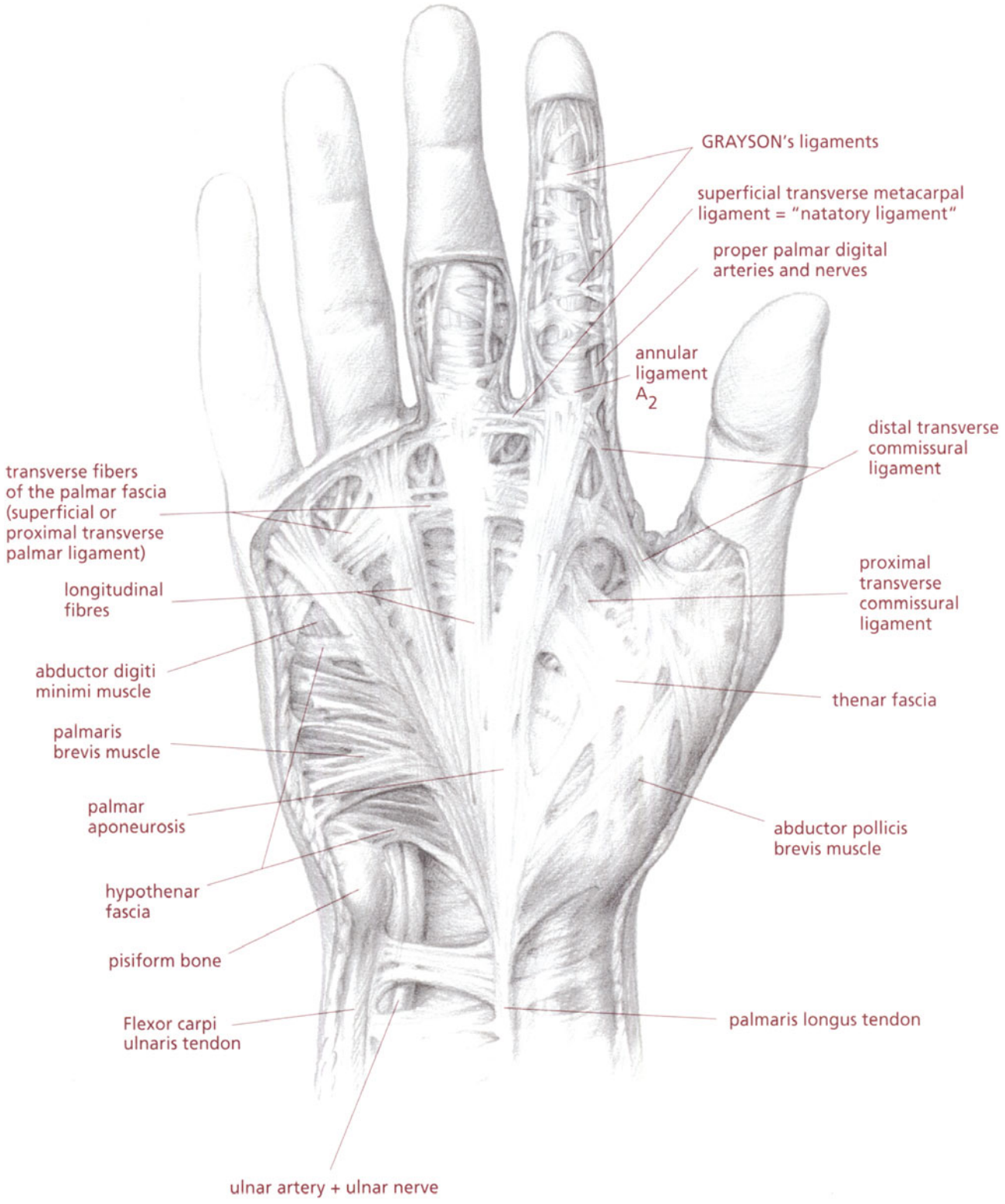


Fig. 18b. The palmar fascial complex comprises five anatomical regions: the radial (thenar) aponeurosis, the ulnar (hypothenar) aponeurosis, central (palmar) aponeurosis, palmodigital, respectively digitopalmar fascia (transition zone), and digital fascia.

aponeuroses: thenar, hypothenar and palmar. The palmar aponeurosis occupies the central portion of the palm between thenar and hypothenar aponeuroses.

9.2.1 Ulnar aponeurosis

The ulnar aponeurosis comprises the fascia overlying the hypothenar muscle. It is a coalescence of fascial structures more distally and ulnarly and it is bound radially by the pretendinous band to the small finger. LANDSMEER (1976) described the ulnar root of the palmar aponeurosis as derived from the "flexor carpi ulnaris-pisiform-hamate column". WHITE (1984) found that the area over the abductor digiti minimi tendon is a meeting place of fascial strands in which tissue joins from different directions: proximal, distal, radial, ulnar, palmar and dorsal. The abductor digiti minimi tendon continues proximally as this muscle's fascial covering. Distally the tendon fibers are in proximity to the digital fascia including lateral digital sheet. Radially the abductor digiti minimi tendon attaches to the palmar plate of the metacarpophalangeal joint. Ulnarly there are vertical fibers that connect the tendinous tissue to the dermis. Palmarly the transverse ligament of the palmar aponeurosis merges with the abductor digiti minimi tendon. Dorsally the abductor digiti minimi tendon is enveloped by the fibers of the sagittal band—as previously described in a study from our institution (RAYAN et al., 1997).

Fine anatomical dissection (RAYAN, 1999) revealed that the hypothenar fascia is an extension of the palmar aponeurosis. This fascia is substantially thinner than the palmar aponeurosis. Vertical fibers that connect this fascia to the dermis are scant, but relatively strong and cannot be divided easily with the back of a surgical knife blade. The ulnar aponeurosis begins proximally at the flexor carpi ulnaris-pisiform junction, extends to the abductor digiti minimi muscle fascia and terminates distally by the abductor digiti minimi fascial coalescence. The palmar aponeurosis is connected indirectly to the flexor carpi ulnaris-pisiform column by the hypothenar fascia over the pisohamate ligament.

The *abductor digiti minimi* cord may originate in the distal palm ulnarly with extension proximally into the muscle fascia, but most often this cord is digital and located within the small finger. The *isolated digital cord* in the small finger is also known as the abductor digiti minimi cord or isolated spiral digital cord as opposed to the typical spiral cord. This cord, which is encountered on the ulnar side of the hand, was described by BARTON (1984) who found it to take origin from the abductor digiti minimi tendon, but it may also arise from the nearby muscle fascia or base of the proximal phalanx (Fig. 20).

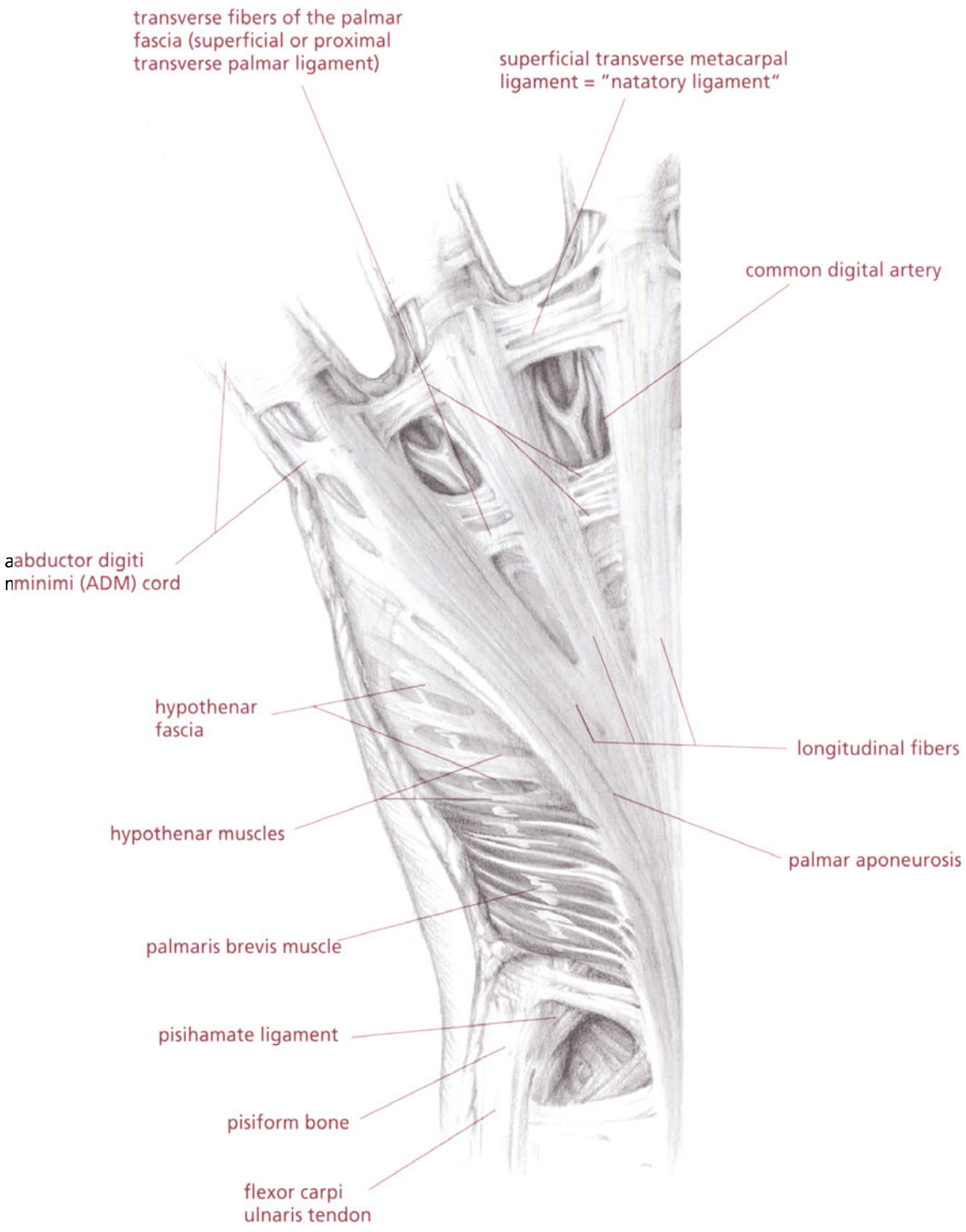


Fig. 18c. The ulnar aponeurosis.

9.2.2 Radial aponeurosis

TUBIANA, SIMMONS and DEFRENNE (1982) studied the fascial anatomy of the radial aspect of the hand and identified four components to the radial aponeurosis: (1) The thenar fascia, which is most radial and covers the thenar muscles. (2) The pretendinous band of the thumb is the smallest fascicle and departs from the radial aspect of the palmar aponeurosis. (3) The distal transverse commissural ligament, which is the continuation of the natatory ligament, the fibers of which are almost perpendicular to the long axis of the abducted thumb (Fig. 19a, b). (4) The proximal transverse commissural ligament of the first web space, which is the radial continuation of the transverse ligament of the palmar aponeurosis. The fibers of the proximal and distal commissural ligaments separate as they travel radially toward their insertion over the thenar fascia. These commissural ligaments have the function of limiting the spread or radial abduction of the thumb. The fascial architecture of the thumb includes the retaining skin ligaments that are retrovascular and analogous to CLELAND's ligament and prevascular which are analogous to GRAYSON's ligament (Fig. 19b).

Fine anatomical dissections (RAYAN et al., 1999) revealed that the thenar fascia blends with the palmar aponeurosis, but it is much thinner and more mobile than the palmar aponeurosis. Very few weak vertical bands connect the thenar fascia to the overlying skin. These are fewer than the vertical bands over the hypothenar fascia. The skin is more mobile over the thenar and hypothenar eminences than over the central area of the palm because the scarcity of these vertical fibers peripherally. The pathologic fascia in Dupuytren's disease on the radial side of the hand has the following characteristics: (1) The proximal commissural cord, which originates from the proximal commissural ligament and causes first web space contracture. (2) The distal commissural cord, which is the diseased distal commissural ligament and can cause thumb-index web space contracture. (3) The thumb pretendinous cord, which originates from thumb pretendinous band, causes metacarpophalangeal joint flexion deformity. (4) The thenar fascial cord, which is uncommon, located along the radial side of the thumb, associated with thumb pretendinous cord and analogous to the hypothenar diseased fascia.

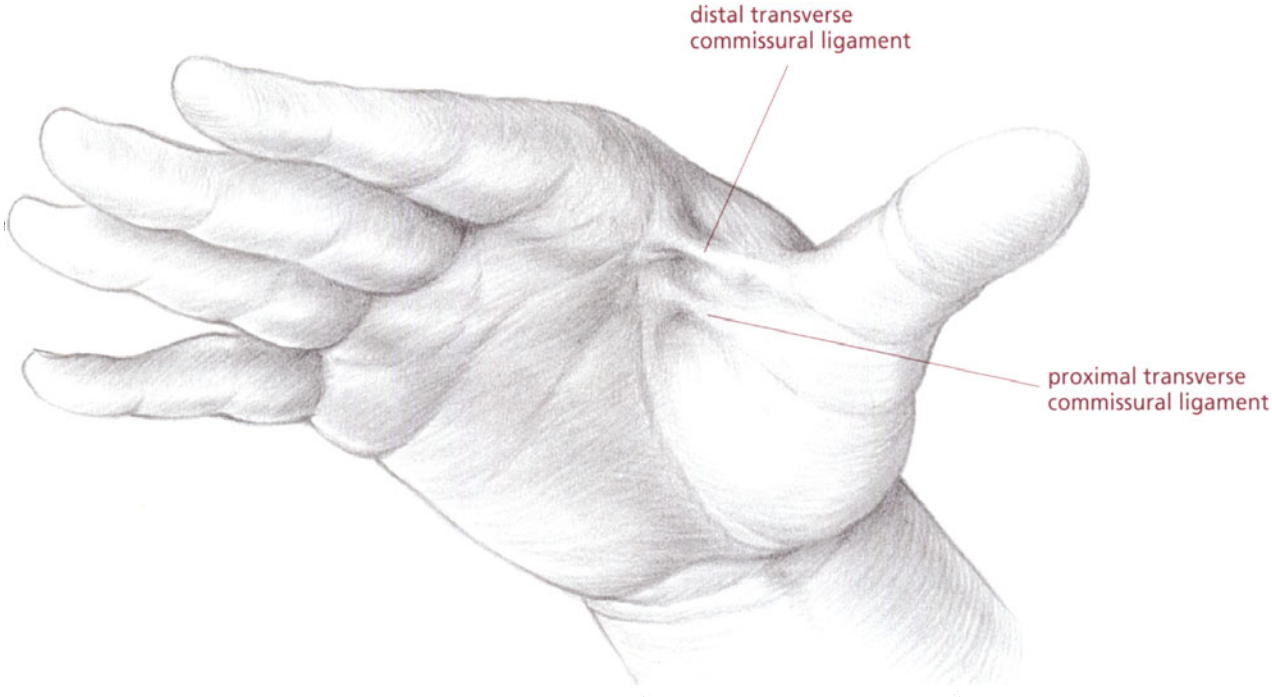


Fig. 19a. Even under normal conditions, the proximal transverse commissural ligament and the distal transverse commissural ligament can be visualized in the first interdigital space, when the thumb is abducted.

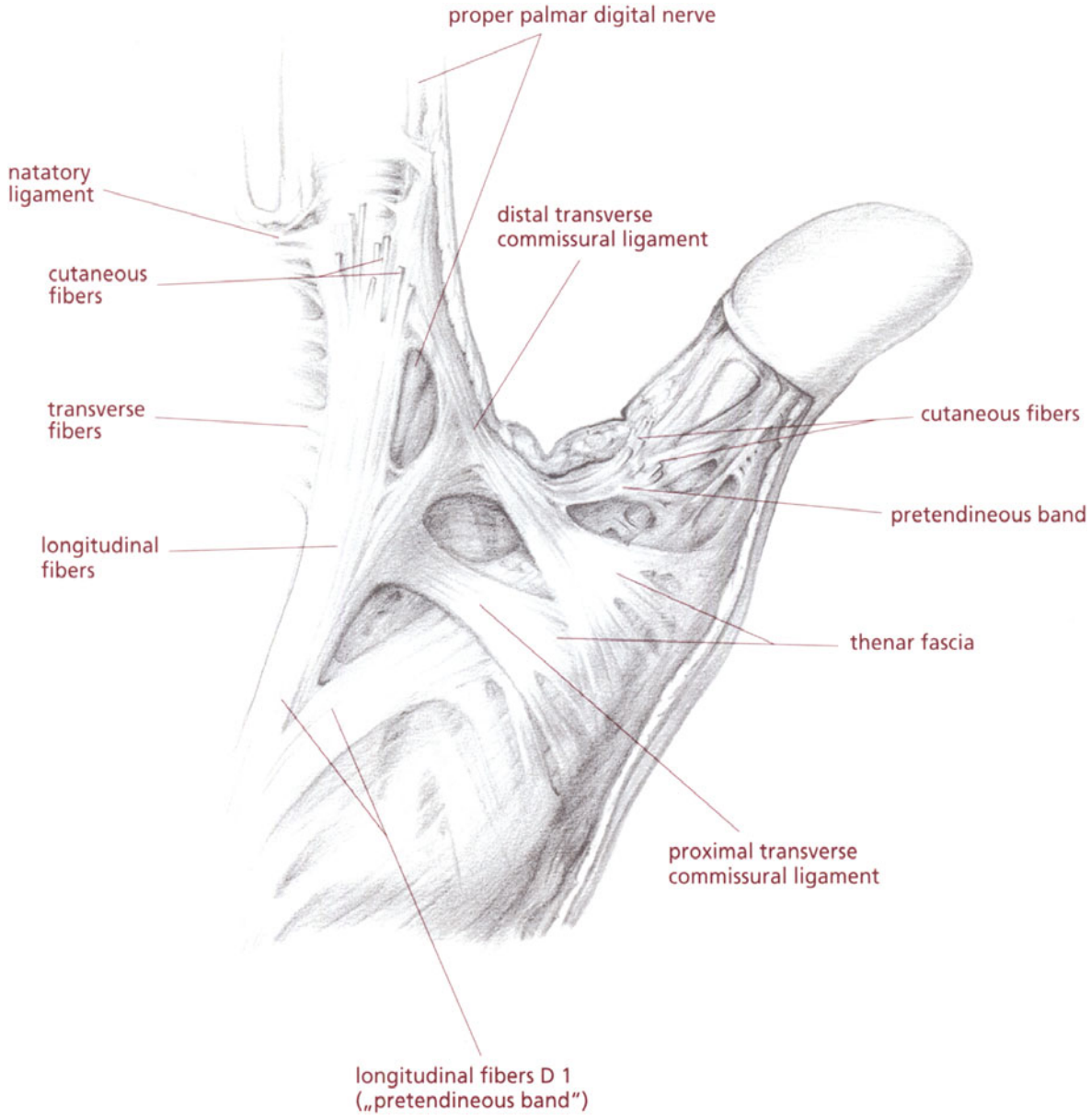


Fig. 19b. Overview of the most important radial fascial systems: pretendinous band with Y-shaped split; strong portion to the index finger, minor portion to the thumb.

9.2.3 Central (palmar) aponeurosis

The palmar aponeurosis is a thick triangular fascial layer and is part of the palmar fascia. It has a proximal apex in the center of the palm and a distal base. When present, the palmaris longus tendon attaches to the proximal apex of the palmar aponeurosis, but the anatomy of the palmar aponeurosis is not different if the palmaris longus is absent. The palmaris longus fibers blend with the palmar aponeurosis and some fibers fan towards the thenar fascia. This probably explains why the palmaris longus tendon becomes very prominent when pinching the thumb against the index and middle fingers or during cupping of the hand and pinching all fingers together while the wrist is flexed. The palmar aponeurosis has a three-dimensional fiber arrangement: longitudinal, transverse and vertical fibers.

9.2.3.1 Longitudinal fibers

ALBINUS (1734) noted the two-pronged bands of longitudinal fibers and DUPUYTREN (1831) described the bifurcated slips and their attachment to the side of the metacarpal heads. GRAPOW (1887) described connections between the palmar aponeurosis and the dermis. LEGUEU and JUVARA (1892) distinguished between pretendinous and intertendinous bands within the palmar aponeurosis, they also found the longitudinal layer of fibers to be continuous with the fasciae of the thenar and hypothenar eminences. Other authors (ANSON et al., 1940; GRODINSKI et al., 1941; KANAVEL et al., 1929) made similar observations: they described the thenar and hypothenar fasciae as an extension of the palmar aponeurosis. On anatomical cross sections GRODINSKI and HOLYOKE (1941) found that the palmar aponeurosis is superficial and adheres to the transverse carpal ligament. They described proximal connecting fibers between the pretendinous bands of the palmar aponeurosis. POIRIER and CHARPIE (1901) observed a distinct separation of the palmar aponeurosis fibers into pretendinous bands (longuettes pretendineuse). FROHSE (1908) described connections between the fibers of the palmar aponeurosis and those of the digital fascia. GOSSET (1985) found connections between the split pretendinous bands and the lateral digital fiber system. MCGROUTHER (1982) used the operating microscope to study the fine anatomy of the distal longitudinal fibers of the palmar aponeurosis and described this longitudinal system as having three different insertions.

The palmar aponeurosis fibers fan out as four bands; one for each digit and each band bifurcates distally and passes on either side of the flexor tendons. A smaller fifth band extends to the thumb. A thin membranous layer of intertendinous fascia connects the three spaces between the four digital pretendinous bands. KALBERG (1935) found variations in the anatomy of these bands with some digits having more than one band, and Y shape bands contributing to two digits. The longitudinal fibers of the pretendinous bands terminate distal to the free edge of the transverse ligament of the palmar aponeurosis and separate into three layers (MCGROUTHER, 1982). The first layer inserts superficially into the dermis, distal to the metacarpophalangeal joint level and midway between the distal palmar and the proximal digital crease. Dupuytren's nodules develop in this area and skin pits result from involvement of these fibers (Fig. 15). The fibers of the second layer pass deep to the natatory ligament and neurovascular bundles and continue towards the digits. MCFARLANE (1974) called these fibers the spiral band of GOSSET (1985). In the digit, these fibers merge with longitudinally oriented fibers named the lateral digital sheet (GOSSET, 1985), and may have attachment to the retrovascular band (THOMINE, 1985). Contracture of these fibers contributes to the formation of a spiral cord. The third deepest layer passes almost vertically on the side of the flexor tendon sheath and metacarpophalangeal joint, and according to McGrouther terminates in the vicinity of the extensor tendon. These fibers were also identified by other authors (KAPLAN, 1938; STACK, 1973; ZANCOLLI, 1979) and named perforating fibers (LEGUEU and JUVARA, 1892). HOLLAND and MCGROUTHER (1997) further identified two different relationships of these fibers to the transversely oriented fibers of the palmar aponeurosis, deep and superficial.

During fine anatomical dissection (RAYAN, 1999), the proximal palmar aponeurosis was found to be very adherent to the underlying transverse carpal ligament. These two structures can be separated only by sharp dissection. This is due to the presence of many connecting fibers that run from the deep surface of the palmar aponeurosis to the transverse carpal ligament. There are many of these strong connecting bands. The palmar aponeurosis is also very adherent to the overlying skin. The thumb pretendinous band is the smallest. The size of the remaining bands is variable, but those of the ulnar two digits are often thicker than the radial ones. The index and middle finger pretendinous bands, however, can be equal or even slightly more developed than those of the ring and small fingers. The pretendinous bands to the index and middle fingers are longer, but sometimes more flimsy than those to the ring and small fingers. Many of the pretendinous coarse fibers insert at an angle into the dermis, not only deep to the palmar creases, but also throughout other areas of the palm. These are different from the numerous small vertical bands superficial to the palmar aponeurosis, which also insert into the dermis.

The pretendinous cord in Dupuytren's disease develops from the pretendinous band and causes

metacarpophalangeal joint flexion deformity. This deformity is the result of the cord's attachment to the skin of the distal palmar crease. This cord often extends distally and continuous with digital cords such as the lateral and central cords. Occasionally the pretendinous cord bifurcates distally and each branch extends to a different digit and forms a commissural Y cord (Gosset, 1985).

9.2.3.2 Transverse fibers

This system of fibers encompasses two separate structures; the natatory ligament, which is located distally, and the transverse ligament of the palmar aponeurosis, which is located proximally. These should be differentiated from the deep transverse inter-metacarpal ligament at the level of the palmar plates (Fig. 20).

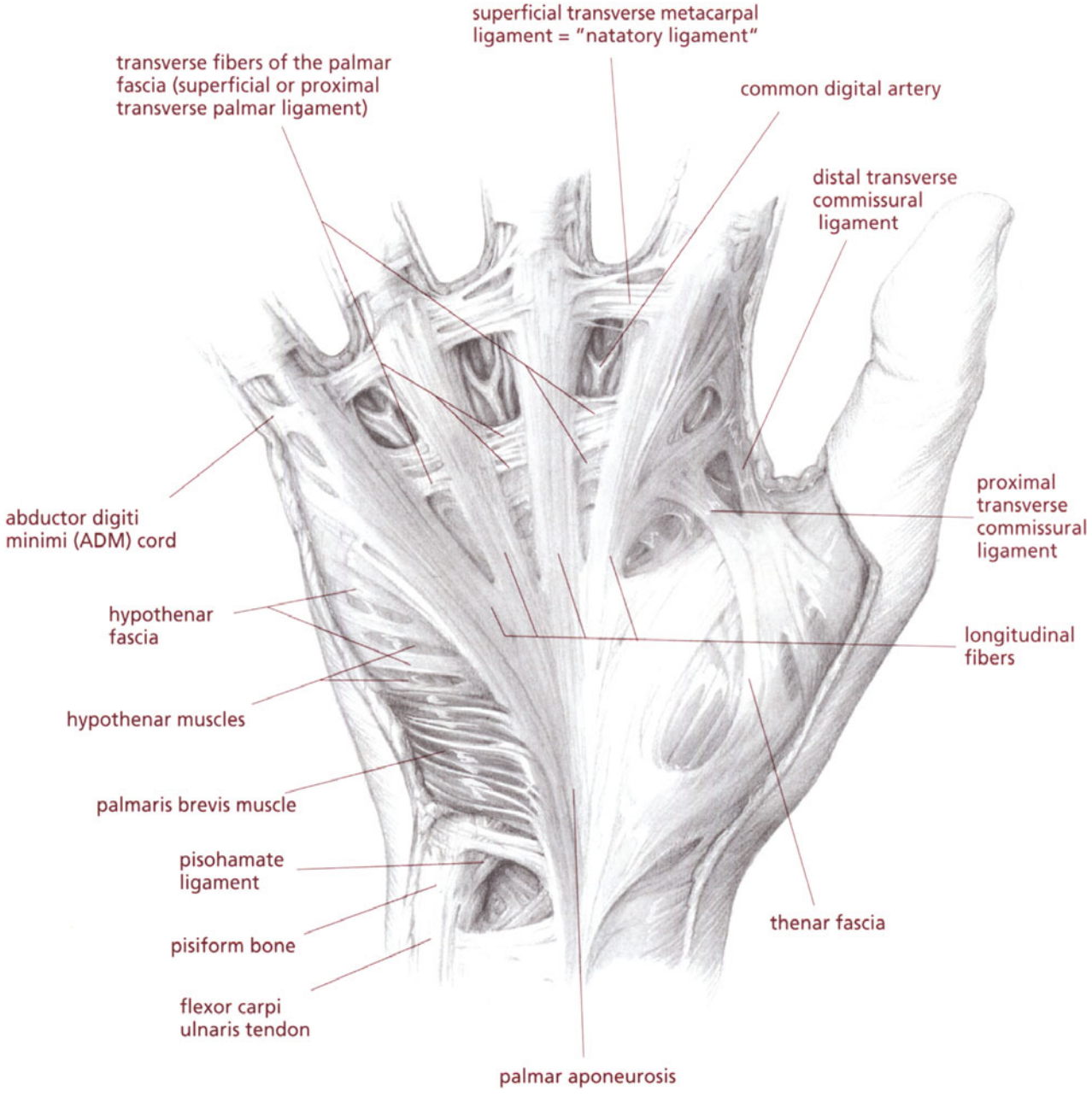


Fig. 20. The natatory ligament extends transversely in the web spaces I-IV of the digitopalmar transition zone. The palmar aponeurosis fans out between the thenar and hypothenar aponeuroses. It consists of longitudinal and transverse fibers. The longitudinal fibers form the pretendinous cords, while the transverse fibers form the TLPA. The TLPA extends below the fasciculi longitudinales.

SKOOG (1948) described the transverse fibers of the palmar aponeurosis underlying the distal palmar crease and appreciated their location deep to the pretendinous bands. LEGUEU and JUVARA (1892) used the term "superficial transverse ligament" to describe these fibers which lie at a deeper plane than the natatory ligament and pretendinous bands. GOSSET (1985) described a "tough band" connecting this ligament to the flexor tendon sheath. The distal border of this ligament also forms arches of commissural spaces over the neurovascular bundles. A retinacular role has been attributed to the distal edge of this ligament deep to the distal palmar crease. On either side, the fibers of this ligament blend with the distal portions of thenar and hypothenar fasciae. Its radial extension is the proximal commissural ligament (TUBIANA et al., 1982). These fibers were observed to be related to the underlying septa of LEGUEU and JUVARA. Although this structure lies deep to the longitudinal fibers and is mostly covered by these fibers, fine dissection showed that it is not separate from the palmar aponeurosis, its fibers occupy the deepest plane of the palmar aponeurosis. Therefore, an appropriate term for these fibers is "transverse ligament of the palmar aponeurosis" (TLPA). The transverse ligament of the palmar aponeurosis is gently convex at its distal border and concave proximally. It extends from the radial aspect of the index to the ulnar aspect of the small finger. The septa of LEGUEU and JUVARA originate from the transverse ligament of the palmar aponeurosis. The main purpose of the transverse ligament of the palmar aponeurosis seems to be that it contributes vertical fibers for the construction of the vertical septa.

The metacarpophalangeal joints' palmar plates are firmly interconnected by deep ligaments, which were named deep transverse inter-metacarpal ligaments (GRATZER et al., 2001). STACK showed that these ligaments have no direct connection with the metacarpal heads, but they lie palmar to the metacarpals and their connection to the metacarpal heads is indirectly through the collateral ligaments. Therefore, they would be more accurately named the "interpalmar plate ligament" (IPPL). These ligaments are located deep to the flexor sheaths and continue with the palmar plates. WEITBRECHT (1742) and ZANCOLLI (1979) made detailed illustrations of this structure. There are three of these ligaments, which are interconnected by the palmar plates, and all appear as one continuous structure.

Fine anatomical dissections (RAYAN, 1999) revealed a soft tissue confluence in the hand, which consists of the sagittal band, septum of LEGUEU and JUVARA, interpalmar plate ligament, palmar plate, and the A_1 pulley of the flexor tendon. The interosseous fascia, the periosteum, and the capsuloligamentous structures of the metacarpophalangeal joint make additional, less substantial contributions to this soft tissue confluence.

The transverse ligament of the palmar aponeurosis of SKOOG (1948, 1967) is spared in Dupuytren's disease. This ligament, however, can be involved in the disease process deep to the longitudinal fibers of the pretendinous band when a vertical cord develops from the septa of LEGUEU and JUVARA. Additionally, this ligament can be involved when its fibers become longitudinally oriented in the first web space i.e. the proximal commissural cord.

9.2.3.3 Vertical fibers

WEITBRECHT (1742) observed fibrous strands that pierce the fat pads. GRAPOW (1887) described connections between the palmar aponeurosis and palmar skin and described multiple attachments of the longitudinal fibers distally through the natatory ligament to the periosteum and through fat to the skin, especially near flexion creases.

Fine anatomical dissections (RAYAN, 1999) revealed that the vertically oriented fibers of the palmar fascial complex occupy two locations: one is superficial and the other deep to the facial complex. The superficial vertical bands are numerous, small, strong, penetrate the adipose tissue and are scattered along the palm. In the central portion, they are grouped around, but not restricted to the palmar crease. They are anchored to the skin, especially in the proximal and mid palm. These bands are most scarce and flimsy over the thenar fascia. They are a little more numerous over the hypothenar area. This explains the limited skin mobility in the mid palm as compared to the skin of the thenar and hypothenar areas. The vertical bands connecting the palmar aponeurosis to the dermis are very abundant, strong and bind the proximal palmar skin to the palmar aponeurosis. Some fibers may run toward the transverse ligament of the palmar fascia on either side of the pretendinous bands, connecting this ligament to

the dermis. Vertical bands were also noticed to connect the natatory ligament to the overlying skin. The second set of vertical bands is deep and connects the palmar aponeurosis and natatory ligament to the fibrous flexor tendon sheath.

The deep vertical fibers constitute three types: (1) The well developed septa of Legueu and Juvara. (2) McGrouther's fibers of the deep layer of the pretendinous band. (3) The small vertical bands that occupy the deep plane beneath the fascia, the fibers of which connect the transverse ligament of the palmar aponeurosis to the flexor tendon sheath.

LEGUEU and JUVARA (1892) described the vertical septa that pass deeply from the palmar aponeurosis, forming fibro-osseous compartments. BOJSEN-MØLLER and SCHMIDT (1974) described these vertical septa as of variable lengths and being best developed distally, where they blend with the deep transverse inter-metacarpal ligament. GOSSET (1985) observed that each of the partitions of LEGUEU and JUVARA has a proximal falciform border at a level midway between distal wrist crease and proximal digital crease. The distal border is at the level of the metacarpophalangeal joint and attaches to the pretendinous bands after their bifurcation (Fig. 21a).

Fine anatomical dissections and intra-compartmental arthroscopic examination (RAYAN, 1999) revealed that each of the vertical septa consists of a well developed fibrous structure which has sharp and very strong proximal border and lies approximately 1 cm distal to the palmar arterial arch (Fig. 21b). Each septum is approximately 1 cm in length and varies depending on the digit,

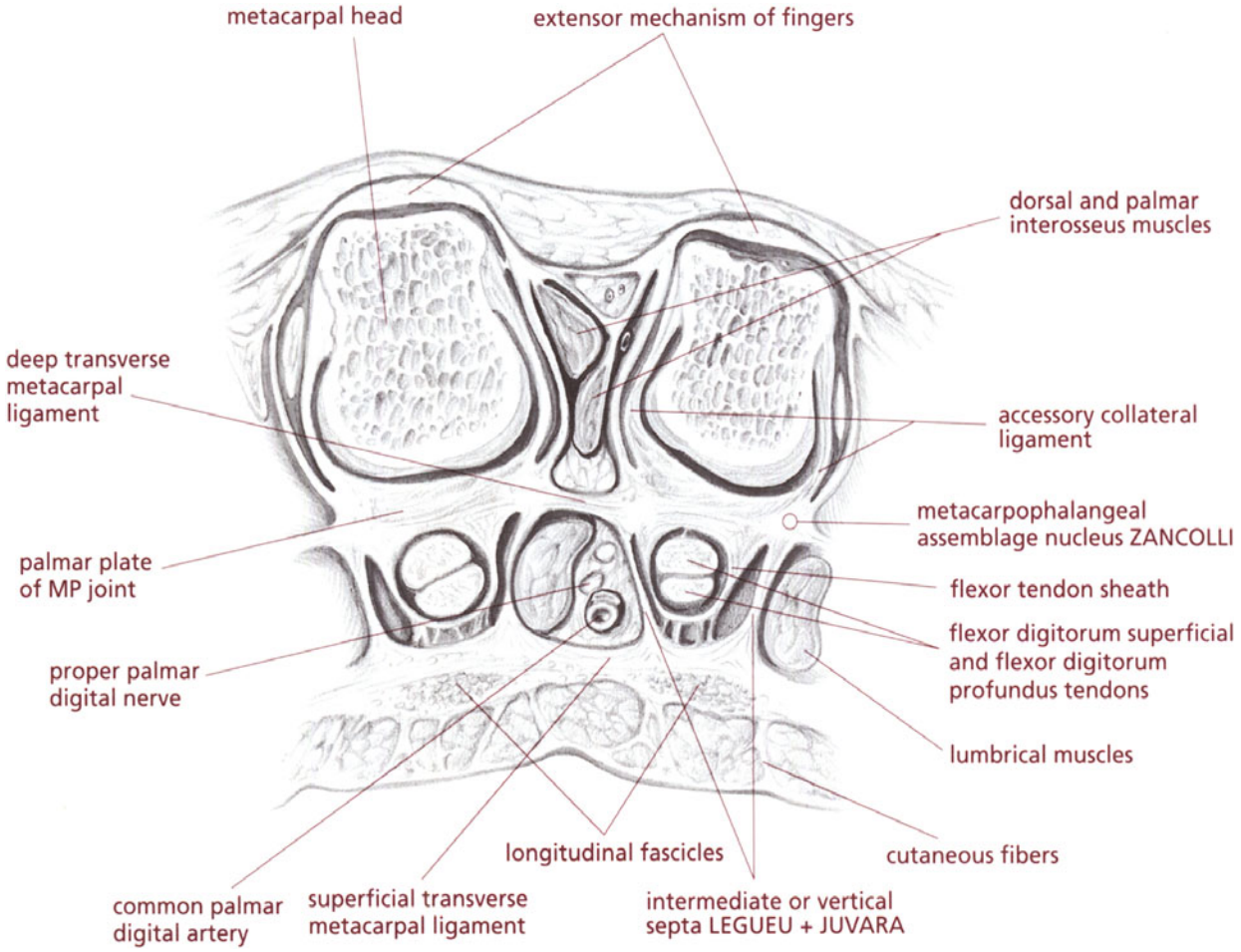


Fig. 21a. Anatomical cross section of two central digits at the level of the metacarpal heads. The following structures can be recognized from palmar to dorsal: palmar skin, vertical skin anchoring system of the subcutaneous tissue, palmar aponeurosis; the keel-shaped intermediate or vertical septa Legueu and Juvara, which derive from the palmar aponeurosis; the palmar plate, interpalmar plate ligament (IPPL) = deep transverse metacarpal ligament (Nomina Anatomica), metacarpophalangeal assemblage nucleus ZANCOLLI, head of metacarpal bones, extensor apparatus with extensor tendon and finally the skin of the dorsum of the hand.

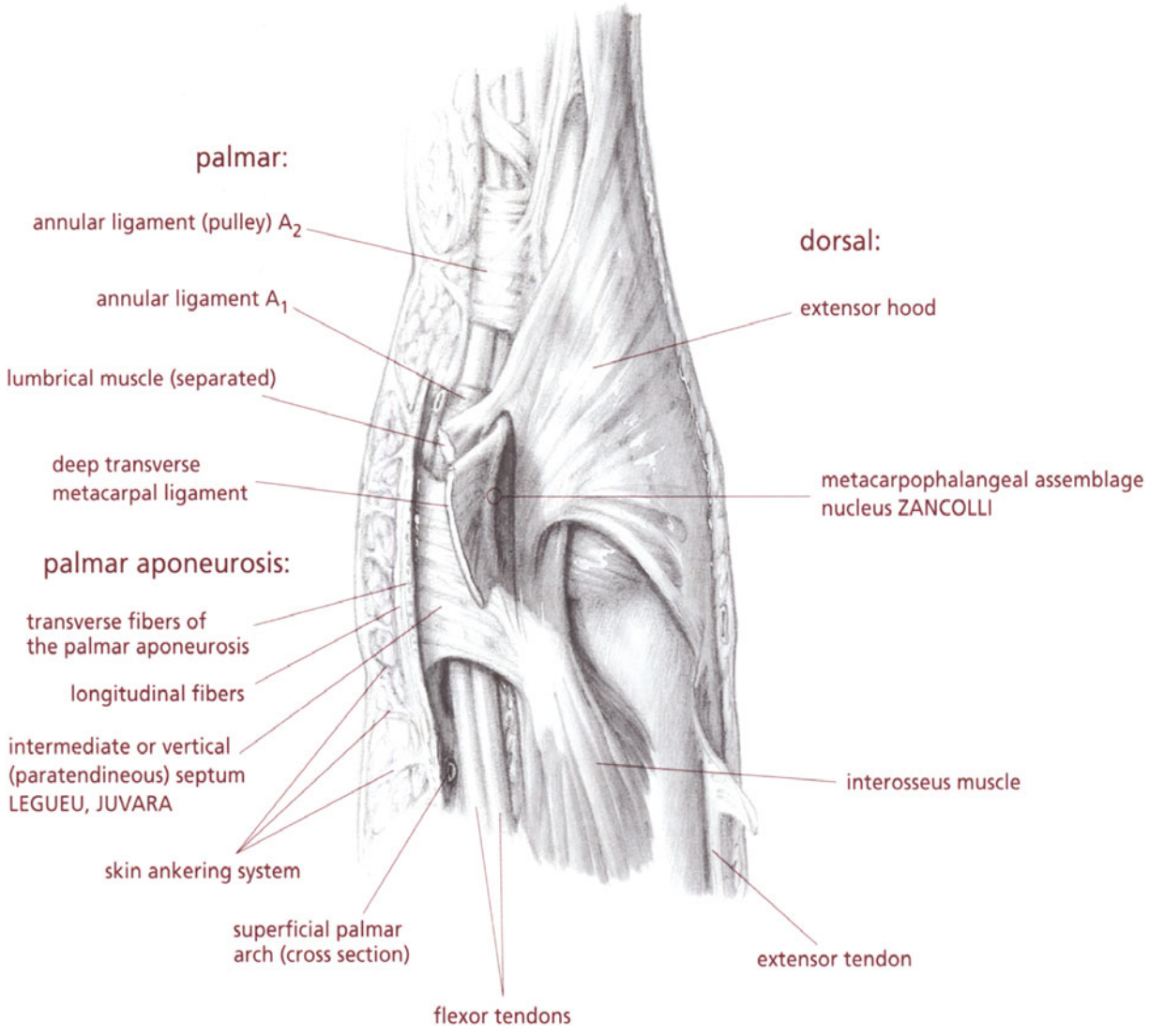


Fig. 21b. Three-dimensional longitudinal section of the metacarpophalangeal region. The same anatomical structures as in Fig. 21a are shown in a sagittal direction. The metacarpophalangeal assemblage nucleus is the mechanical epicenter as well for the paratendinous septa Legueu and Juvara as for the palmar plate, the deep transverse metacarpal ligament and the A_1 pulley.

from 8–12 mm, but it extends further distally and becomes thin and membranous. The proximal border of the vertical septum is proximal to the level of the metacarpal neck, and its distal border is at the metacarpal head and overlaps with the proximal portion of the palmar plate. The vertical septa are located deep to and originate from the inferior surface of transverse ligament of the palmar aponeurosis and extend deeply to attach into the interosseous muscle fascia proximally and interpalmar plate ligament distally and blend with the soft tissue confluence. The most peripheral two septa insert into the intrinsic muscle fascia dorsally. There are eight septa one on each side of the flexor tendon sheath. These are also organized as two septa in the vicinity of each of the three digital web spaces (second, third and fourth), one radial to the index and the last is ulnar to the small finger (Fig. 36). These eight vertical septa form seven fibro-osseous compartments, or partitions, four containing the flexor tendons and three containing the common digital neurovascular bundles of the three web spaces along with the lumbrical muscles. The radial neurovascular bundle of the index finger and the ulnar neurovascular bundle of the small finger are not contained within compart-

ments. Each vertical septum separates the flexor tendon partition from the neurovascular bundle-lumbrical partition. The vertical septum appears to continue with the fibrous flexor tendon sheath, but the latter is contained within the vertical septa. When the arthroscope is introduced into the flexor tendon partition, the flexor tendons can be visualized distally to the level of the proximal interphalangeal joint. When the arthroscope is introduced into each of the neurovascular bundle-lumbrical partition it will stop at a dead end in each of the three web space areas.

A *vertical cord* is uncommon and appears as a short thick diseased tissue, bifurcating from the pretendinous cord and extending deeply in between the neurovascular bundle and flexor tendon fibrous sheath. It inserts into the soft tissue confluence. This cord represents either the diseased vertical fibers of McGrouther or most likely the diseased septa of Legueu and Juvara. This cord may contribute to painful stenosing tenosynovitis. Gosset described similar diseased tissue extending into the intertendinous partition.

9.2.4 Palmo-digital fascia

The palmo-digital area anatomy is difficult to comprehend because of the convoluted and intertwined nature of its soft tissue structures. Gosset (1968, 1985) provided a detailed description of the anatomy of the web-digital area. Each bifurcated pretendinous band spirals and twists on its axis nearly 90° and the peripheral fibers run vertically and become adherent to the metacarpophalangeal joint capsule (spiral bands of Gosset). Some of these fibers fuse with the distal portion of the septa of Legueu and Juvara and continue distally deep to the neurovascular bundle and natatory ligament to emerge distal to the natatory ligament and continue as the lateral digital sheet. The lateral digital sheet therefore has deep and superficial contributions from the spiral band and natatory ligament. In the palmo-digital (synonymous: digitopalmar) region, the neurovascular bundle is entrapped in this confluence of aponeurotic fibers. The fibers of the natatory ligament run primarily in a transverse plane, but the distal fibers form a U and run longitudinally towards the lateral digital sheet (LDS) along the side of the digit (Fig. 21c, d).

The *spiral cord* is named as such because of its relationship to the neurovascular bundle. The cord may have a proximal connection in the palm (typical) or it may be confined to a digit (isolated). The typical spiral cord has four components of origin; pretendinous band, spiral band, lateral digital sheet, and Grayson's ligament (Figs. 22a, b and 28). The presence of a spiral nerve is usually predicted by the presence of proximal interphalangeal joint contracture and an interdigital soft tissue mass (Ulmas et al. 1994).

A spiral cord may originate proximally from the lumbrical tendon or it may attach distally to the opposite "reverse spiral cord" (McFarlane, 1985). Most frequently, the isolated spiral digital cord affects the small finger where it is also known as the abductor digiti minimi cord. It arises from the abductor digiti minimi musculotendinous junction proximally and takes the same course distally as the typical spiral cord (Fig. 32a–32c).

Grapow (1887) alluded to the natatory ligament, and standard anatomy texts refer to this ligament as the "superficial transverse metacarpal ligament". The fibers of the natatory ligament pass from side to side at the web spaces and some fibers pass distally on either side of the digit. The natatory ligament also radiates to the first web space and proximal thumb crease as the distal commissural ligament. Thomine (1985) considered the natatory ligament to be one structure with a straight proximal border extending from the index to the small finger. Fibers from its distal border correspond to the web spaces, contribute to the digital fascia and continues lateral to the neurovascular bundle.

Fine anatomical dissection (Rayan, 1999) showed the natatory ligament to have a well-defined proximal border, which migrates palmarly away from neurovascular bundles, becomes more pronounced and tight when the index and small fingers are fully abducted. The width of the natatory ligament from the web space to its proximal border is approximately 1 cm, and the common digital neurovascular bundle bifurcates proximal to the natatory ligament, especially the nerve, which often bifurcates proximal to the artery. The natatory ligament has one continuous proximal border, but distally there are three components to the natatory ligament; one for each of the second, third, and fourth web spaces, in addition to its radial extension to the first web space. In these three areas, the natatory ligament provides longitudinal fibers to adjacent digits, which contribute to the formation of the lateral digital sheet. The

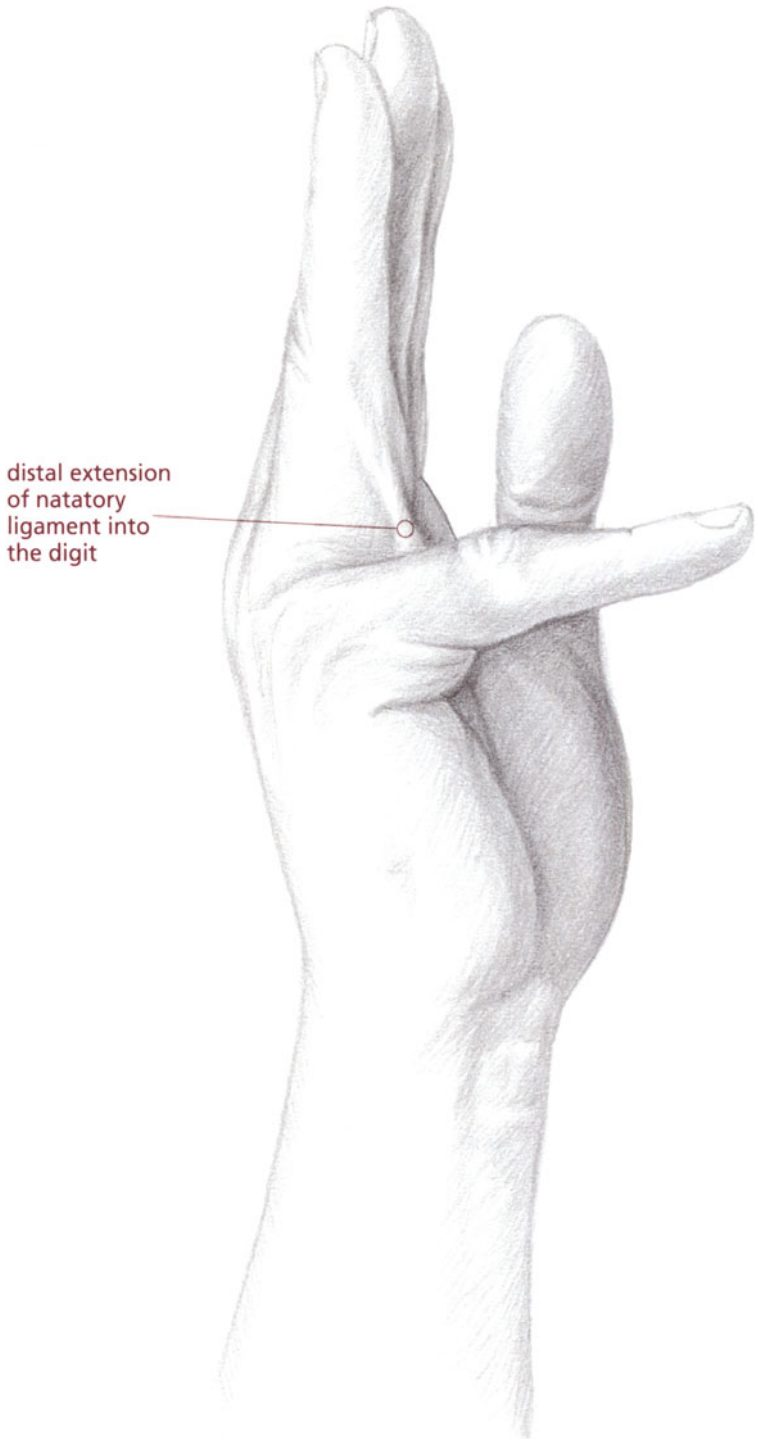


Fig. 21c. The digital extension of the natatory ligament. The band is most easily visible if the fingers are abducted, with one finger flexed at the MP joint and the other one extended.

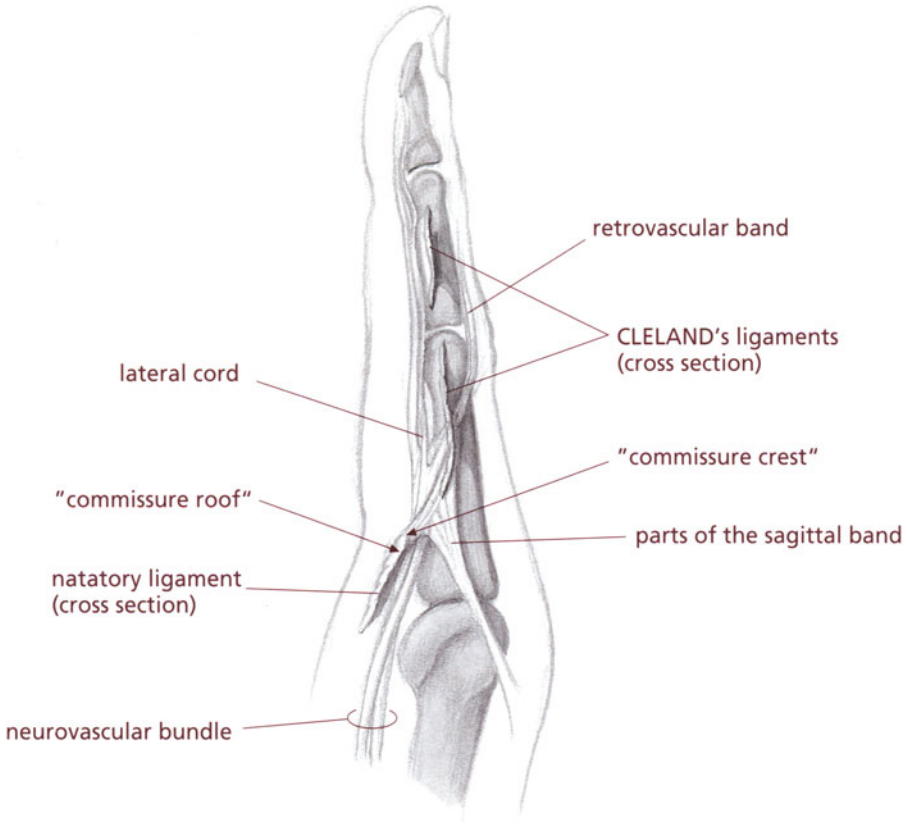


Fig. 21d. The lateral cord is a centro-lateral fascial condensate of the tubular digital sheath and does not cause a contracture in itself. The palmar parts of the natatory ligament form the "commissure roof" and the dorsal parts jointly form the "commissure crest." Both parts merge at the level of the middle phalanx and contribute to the retrovascular ligament. Because of the link between the natatory ligament and parts of the retrovascular ligament, a flexion deficit can result in individual cases.

fibers of the natatory ligament are adherent to the skin and continuous along the width of the hand. Immediately proximal to the natatory ligament, there is an abundance of adipose tissue. The fibers of the natatory ligament are readily visible in hands with less subcutaneous fat.

The *natatory cord* develops from the distal fibers of the natatory ligament and converts the U shape web space fibers into a V shape and causes limitation of digital abduction. The cord extends along the dorso-lateral aspect of the digits and can be best detected by passively abducting the digits and at the same time flexing one digit and extending the other at the metacarpophalangeal joints. This maneuver allows the extension of the natatory cord into the digit to become prominent (Fig. 21c).

9.2.5 Digital fascia

The digital fascial structures were classified by MILFORD (1968) into two types: retaining ligaments of the skin and those of extensor mechanism. The retaining ligaments of the extensor mechanism are the triangular ligament, sagittal band, transverse retinacular ligament, and oblique retinacular ligament of Landsmeer. GRAYSON's and CLELAND's ligaments retain the digital skin during flexion and extension. GRAYSON (1941) described a sheet-like fascia that varies in thickness from one individual to another. The ligament passes from the fibrous tendon sheath in the midline to the skin and crosses superficial to the neurovascular bundles. CLELAND (1878) described the ligament which is deeper and thicker than Grayson's ligament and which arises from the side of the phalanges and runs obliquely toward the skin deep to the neurovascular bundle. Other components of the digital fascia are the fibers of the lateral digital sheet, which are on either side of each digit lateral to neurovascular bundles. This sheet receives fibers from the natatory ligament

superficially and the spiral band deeply. The spiral band consists of fibers that surround the neurovascular bundles and reach the lateral digital sheet deep to the neurovascular bundle (Fig. 22a, b).

The retrovascular fascia or band was described by THOMINE (1985); and this consists of longitudinally oriented fibers dorsal to the neurovascular bundle. He regarded the digital fascia as a circular sheath enclosing bone and tendons and splitting anteriorly to enclose the neurovascular bundles. This elliptical neurovascular sheath, which he identified on histologic sections, has a dorsal condensation of fibrous tissue named the "retrovascular band". Thomine described this band as a longitudinal structure, which continues proximally with the natatory ligament and has points of fixation to the skin superficially and to the skeleton deeply, i.e. to the base of the proximal phalanx and interphalangeal joint. The retrovascular fibers may also be located dorsomedially, as Cleland's ligament occupies a more dorsal position. GOSSET (1985) doubted the existence of a cylindrical fascial sheath extending throughout the entire length of the digit.

MCFARLANE (1985) believes that Cleland's ligaments, transverse retinacular ligament, and oblique retinacular ligament of Landsmeer are not involved in Dupuytren's disease. If Cleland's ligaments are involved, this may occur in conjunction with the diseased retrovascular fibers. Grayson's

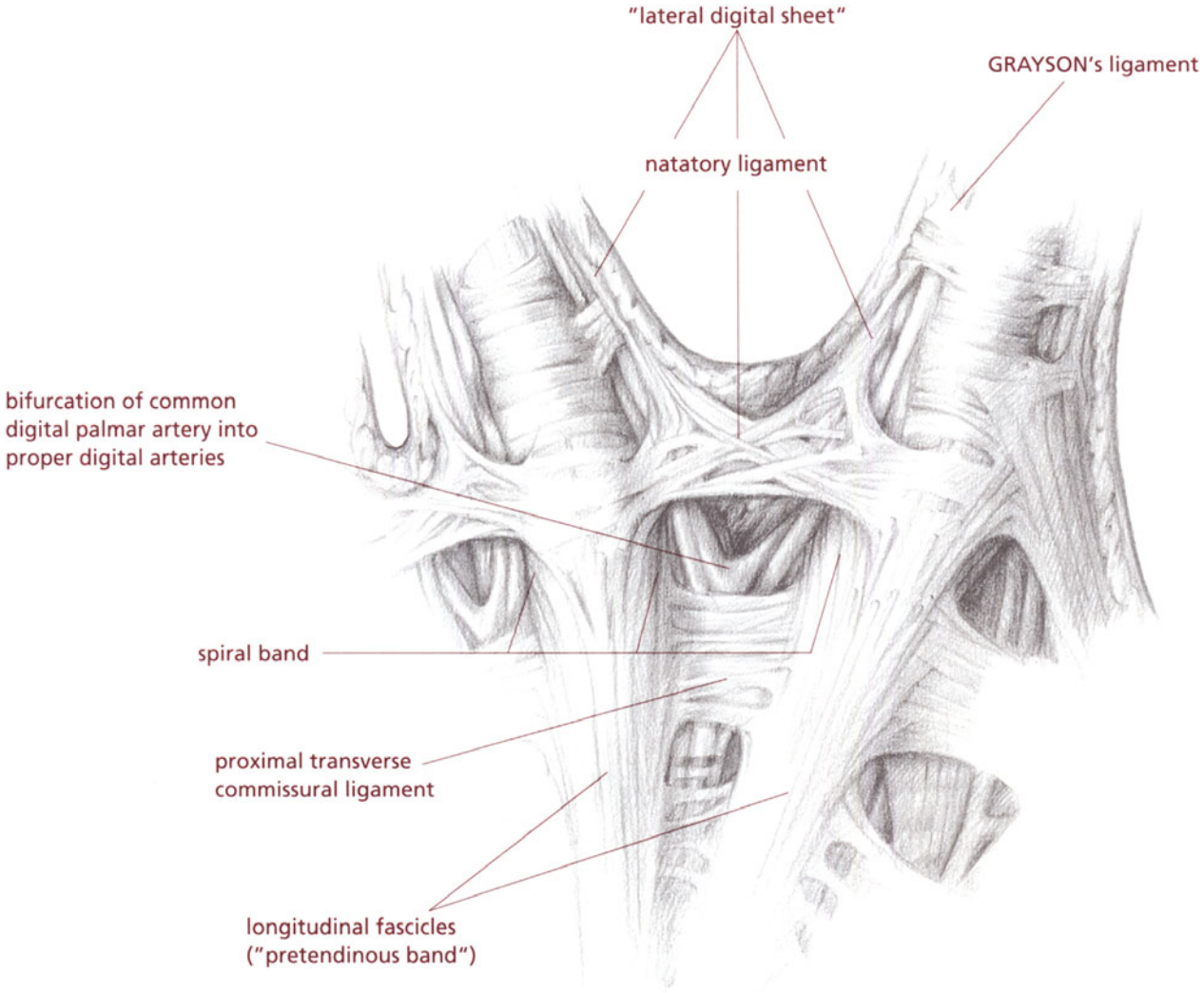


Fig. 22a. The digitopalmar continuum (= palmar fascial complex), which forms the fascial link between palmar and digital fascias (transition zone).

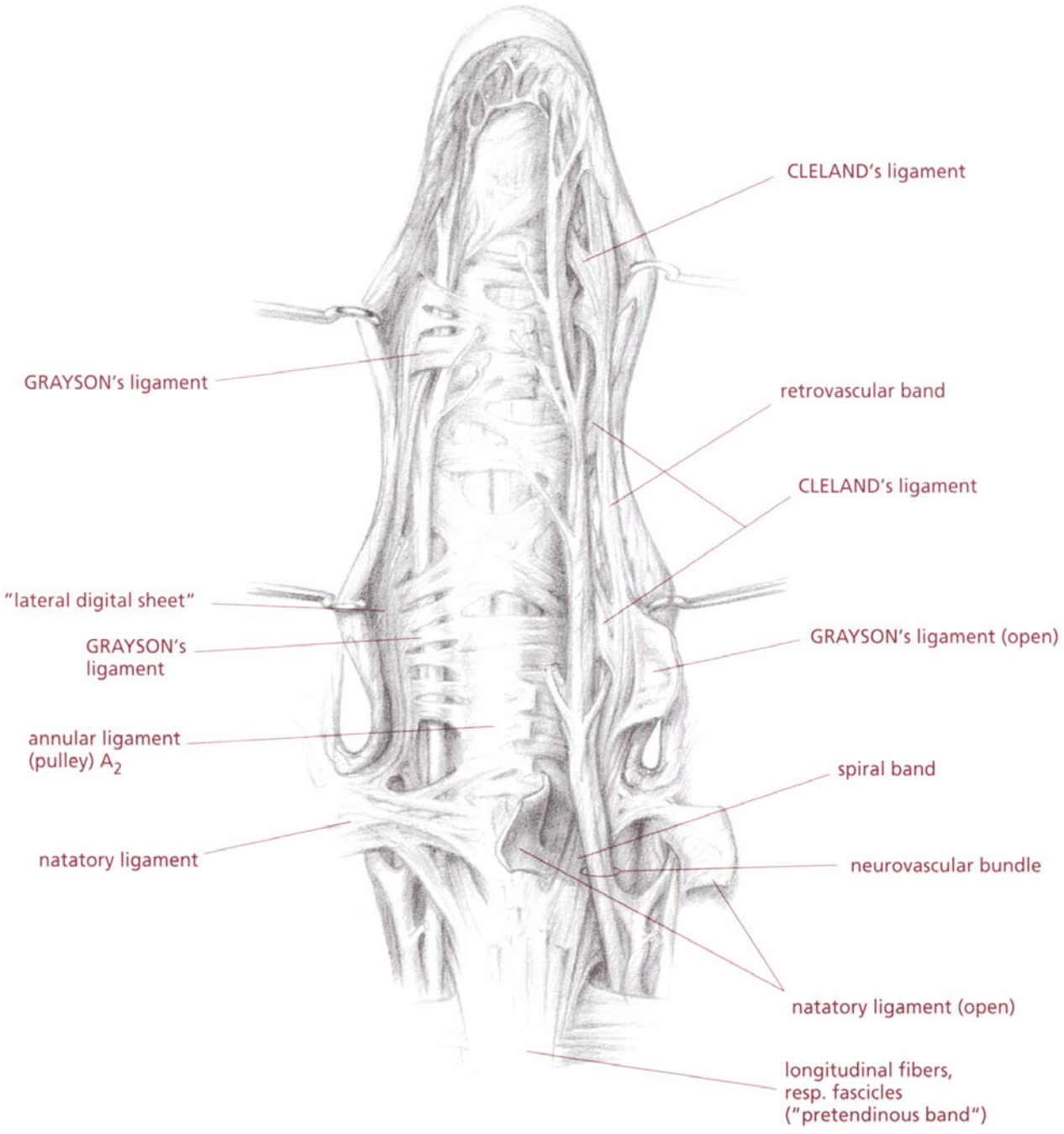


Fig. 22b. Digital portion of the pre-existing fascial architecture.

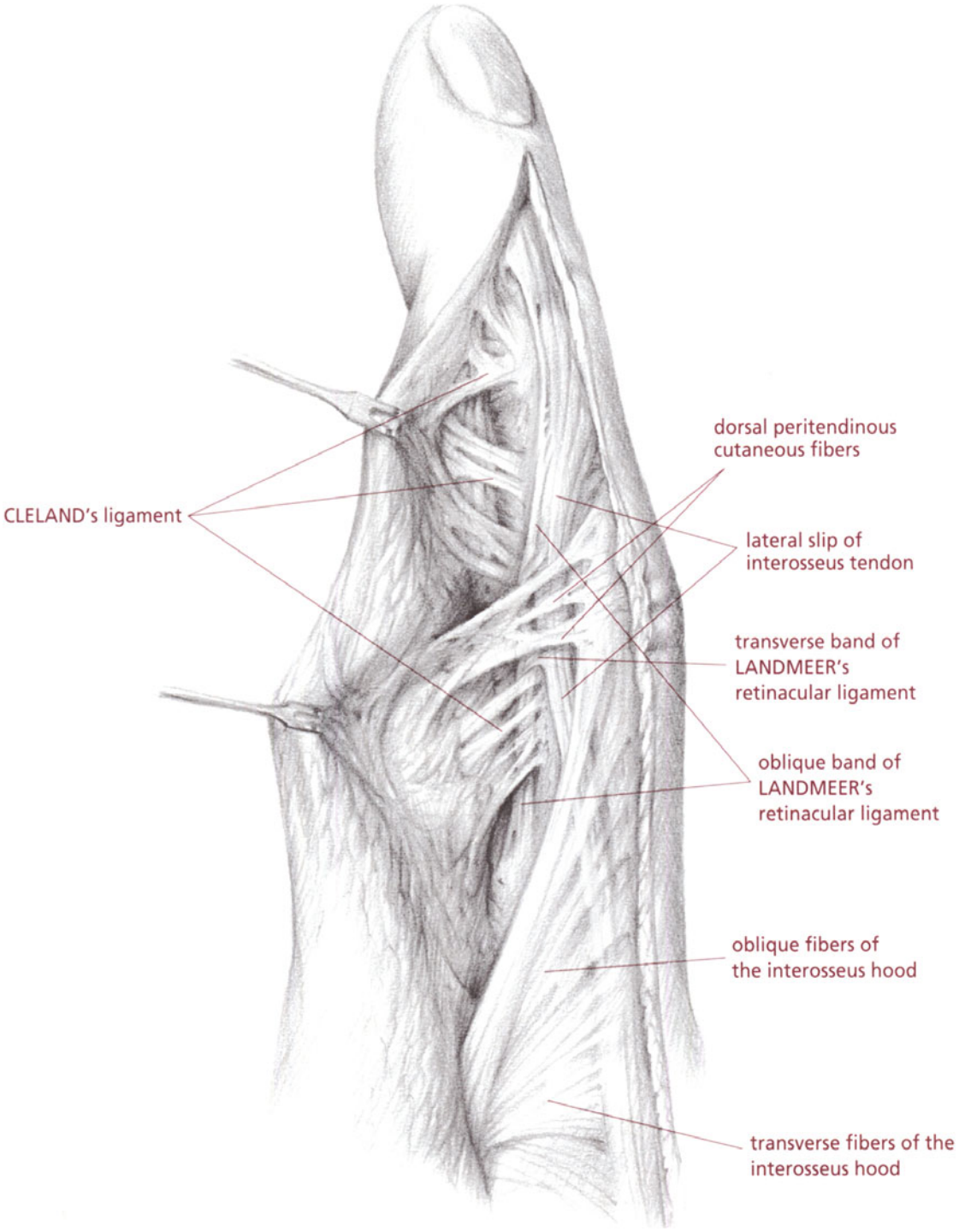

dorsal peritendinous cutaneous fibers
CLELAND's ligament
lateral slip of interosseus tendon
transverse band of LANDMEER's retinacular ligament
oblique band of LANDMEER's retinacular ligament
oblique fibers of the interosseus hood
transverse fibers of the interosseus hood

Fig. 22c. The extensor and retaining ligaments of the digitopalmar continuum (= palmar fascial complex).

ligament becomes diseased as part of the central, spiral or lateral cords.

The *retrovascular cord* according to McFarlane (1985), develops from the retrovascular band of Thomine, and diseased tissue may be present deep to the neurovascular bundle. The diseased retrovascular tissue does not cause proximal interphalangeal joint contracture by itself, but if it is not removed, full correction of proximal interphalangeal joint contracture may not be obtained and residual joint contracture may result. The retrovascular cord can also cause distal interphalangeal joint contracture.

The *central cord* is often an extension of the pretendinous cord into the digit and may develop from the superficial fibrofatty tissue. It courses in the midline between the neurovascular bundles in proximity to the dermis. It attaches to the flexor tendon sheath or the periosteum of the middle phalanx on one side of the digit. In certain advanced cases, when the central cord causes severe joint contracture, it may adhere to Grayson's ligament, which becomes diseased, and displace the neurovascular bundle slightly to the midline.

The *lateral cord* is the diseased lateral digital sheet. It is often associated with a pretendinous cord, but infrequently present without a pretendinous cord. McFarlane (1985) observed that this cord is often associated with a natatory cord. It attaches to the skin or to the tendon sheath through Grayson's ligament. It causes flexion contracture of the proximal interphalangeal joint and sometimes the distal interphalangeal joint. This cord can displace the digital neurovascular bundle by its volume towards the midline (McFarlane, 1985).

10 Classification

A classification into stages is necessary to establish indications and to assess the treatment outcome.

For practical reasons, the grading system by TUBIANA et al. (1961, 1986, 1999) is the most popular. It primarily assesses the extension deficit. The inaugurator subdivides the hand into five digitopalmar segments. Each segment consists of a finger including its palmar zone. For scoring purposes, the first interdigital space counts as part of the thumb segment. For each segment, the palmar and digital lesions are assigned to a particular stage; the score of that particular longitudinal segment also reflects the stage. Each stage corresponds to a stepchange or an extension deficit of 45 degrees of a finger or segment. The total deformity or contracture score of each finger is determined as the sum of the extension deficit of all three digital joints (metacarpophalangeal, proximal and distal interphalangeal joints). If there is hyperextension of the distal interphalangeal joint, its degree is added to the total sum of the contracture (Figs. 23a–23e and 24a–24d). The total range of motion, respectively the lack of extension for individual fingers, can thus be 0–200 degrees in severe contracture, if it is graduated from the distal interphalangeal joint up to the palmar zone. For the thumb with its two joints (metacarpophalangeal and interphalangeal joint) the deformities can be 0–160 degrees. The degrees of severity or stages of flexion contracture of the fingers and their palmar segments can be divided into six stages, as follows.

Stage 0:	no extension deficit, no lesion (nil points)
Stage N:	The suffix N describes a nodule, but without contracture (just 0.5 points)
Stage 1:	The sum of the extension deficit of all three finger joints is equivalent or less than 45 degrees
Stage 2:	Total contracture of 46–90 degrees
Stage 3:	Extension deficit reaches 91–135 degrees
Stage 4:	Total contracture reaches more than 135 degrees

TUBIANA writes that the adduction contracture of the first web space corresponds to gradual loss of abduction of 15 degrees for each stage (Fig. 25a–25d). This results in:

Stage 0:	Neither nodule nor loss of abduction
Stage N:	Nodule
Stage 1:	Abduction angle 45–30 degrees
Stage 2:	Abduction angle 29–15 degrees
Stage 3:	Abduction angle 14-0 degrees

Therefore, the subtotal of the extension deficit of all fingers can reach a maximum of 20 points, and the subtotal for the limited abduction of the thumb can reach a maximum of 3 points.

Consequently, the maximal TUBIANA point score is 23.

The letter "D" (digit) in addition to the grading indicates that the lesion is spreading primarily on the fingers. The letter "H" indicates that the distal phalanx is hyperextended, whereas "A" stands for an amputated finger. Absence of an extension deficit is signified by a minus sign ("–").

RUDIGIER (1987) criticized the above grading system, which determines the extent of the mobility impairment but does not distinguish selectively. He criticized the fact that no provisions were made to document the condition of the overlying skin or any impairment of the neurovascular bundle. He also wanted a classification that reflects the relief and topography.

MILLESI (1981), however, thought that the clinical grading system of TUBIANA and MICHON (1961) did justice to clinical demands. Changes in Dupuytren's disease could be documented by suffixes

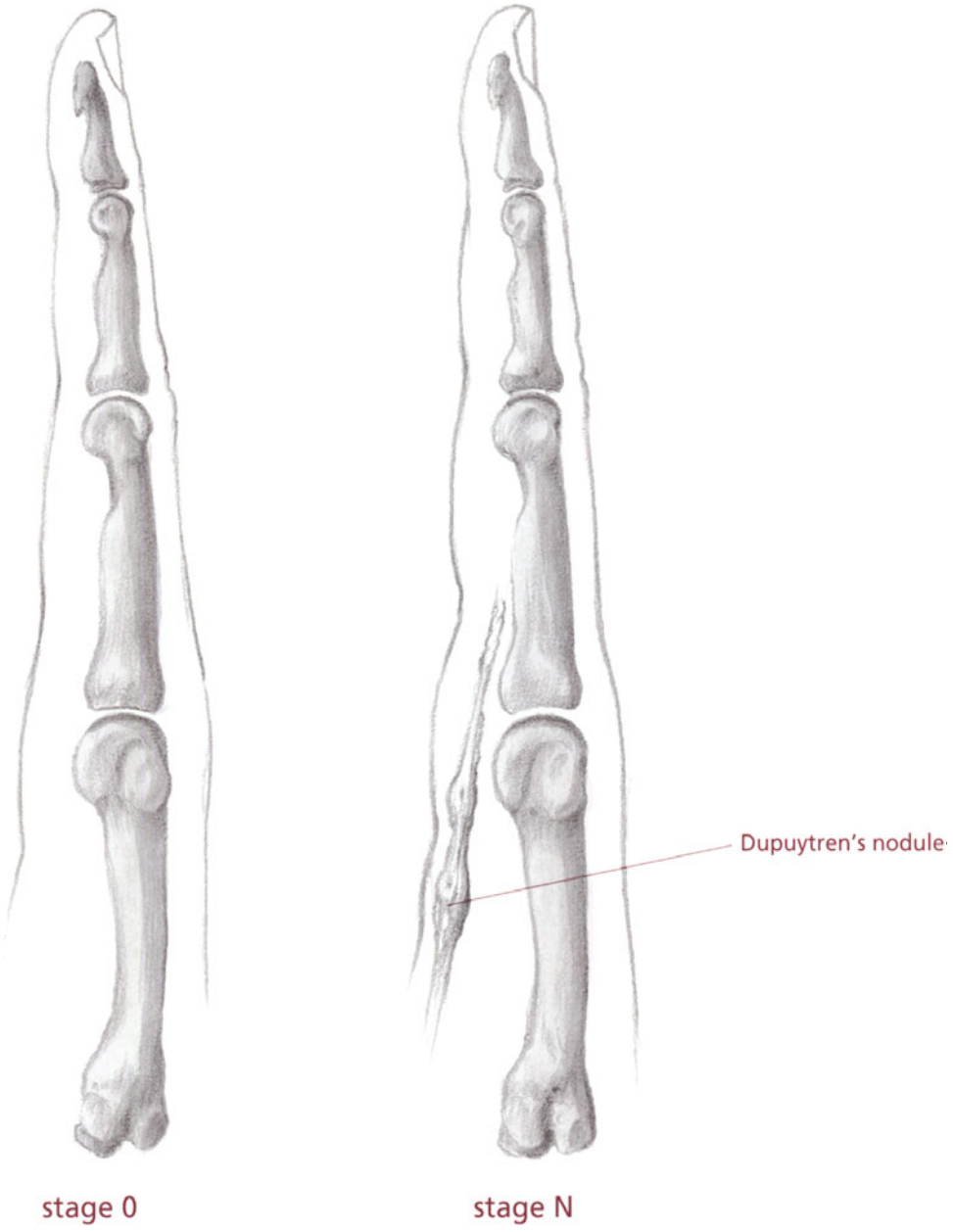


Fig. 23a. Tubiana's classification of the extension deficit of a finger ray differentiates between the following stages: stage 0: no lesion, no extension deficit (no point to be allocated). Stage N (nodular): with palpable nodule, but without contracture (only 0.5 points to be allocated).

added to the individual stages. MILLESI suggests using "F" (fusion), which stands for ankylosis or fusion, "L" (limitation), which stands for flexion deficit, and "R", which stands for relapse.

No universal grading system can encompass the multiple expressions and variations of Dupuytren's disease. Repeated endeavors have been made to use a descriptive classification system that also encompasses criteria such as alcohol misuse, epilepsy, young age, etc. (REBELO et al., 1995). WOODRUFF et al. (1998) conceived such a grading system, which could be used to predict the expected time of the actual operation.

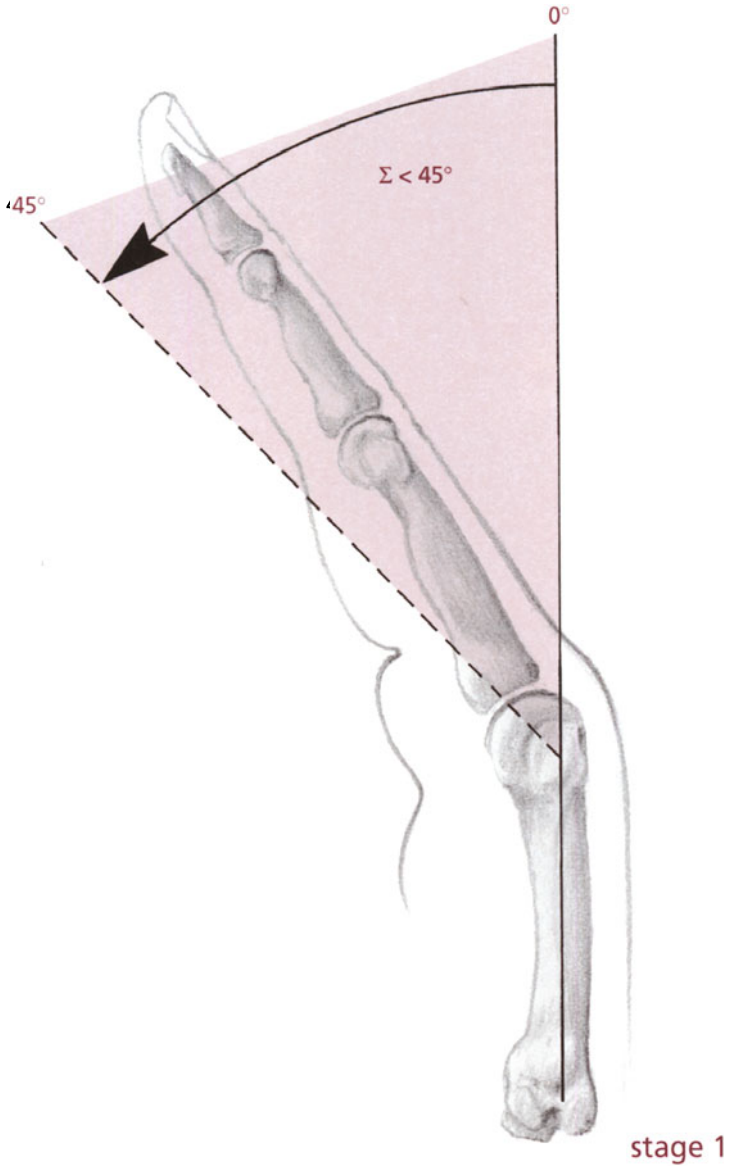


Fig. 23b. The sequential contracture stages reflect an increasing lack of extension in all digital joints in steps of 45 degrees. Stage 1: total contracture of 0–45 degrees.

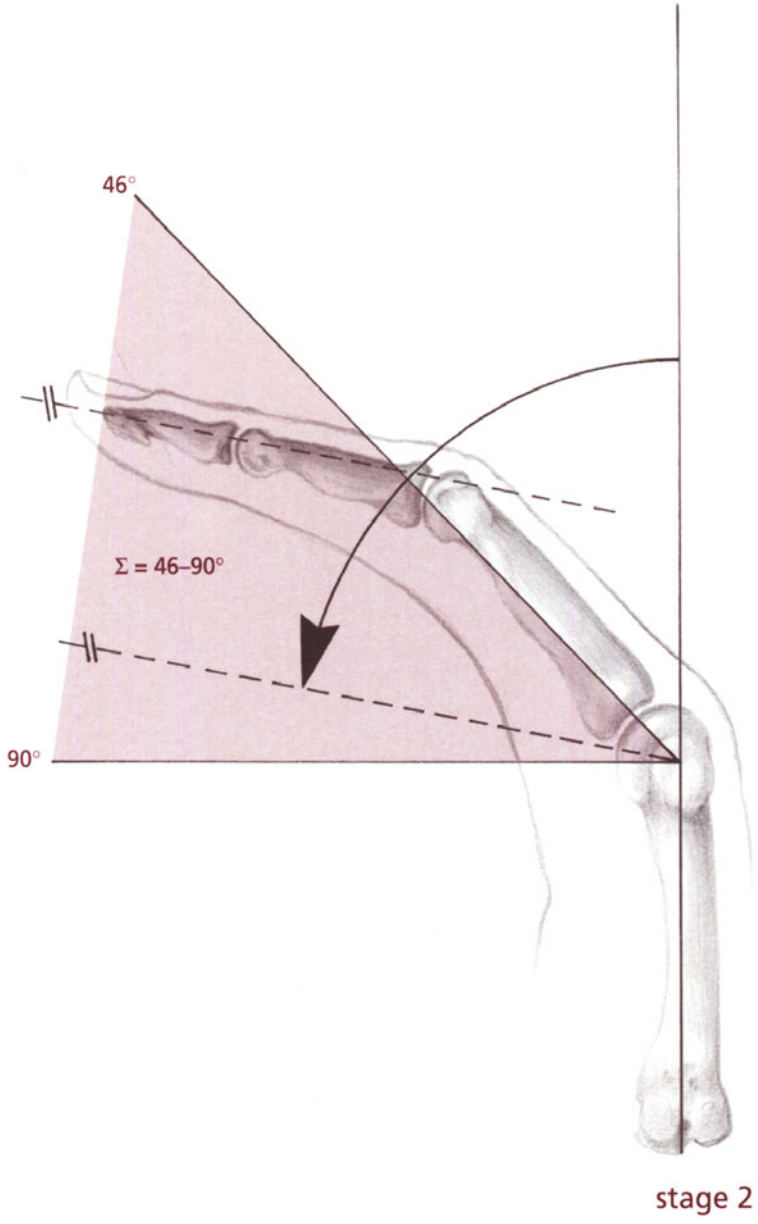


Fig. 23c. Stage 2: global contracture of 46–90 degrees.

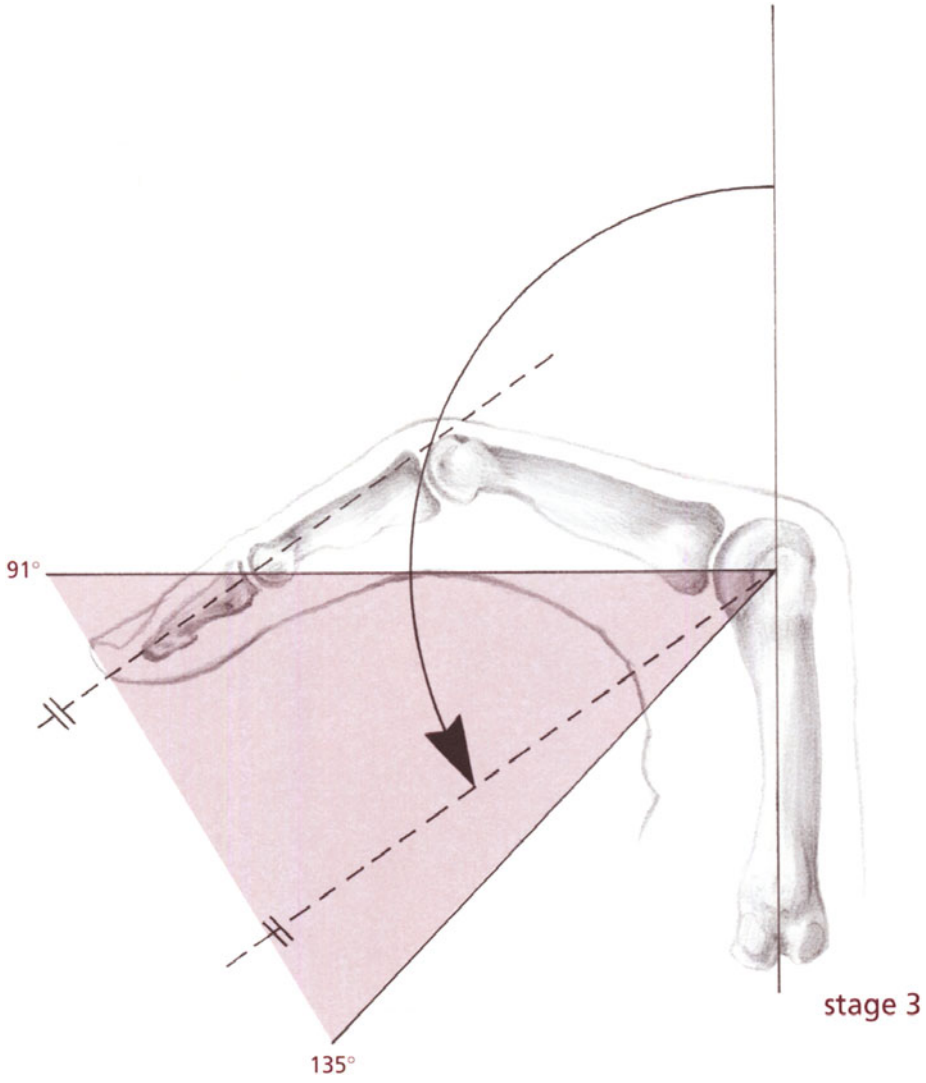


Fig. 23d. Stage 3: Extension deficit of 91–135 degrees.

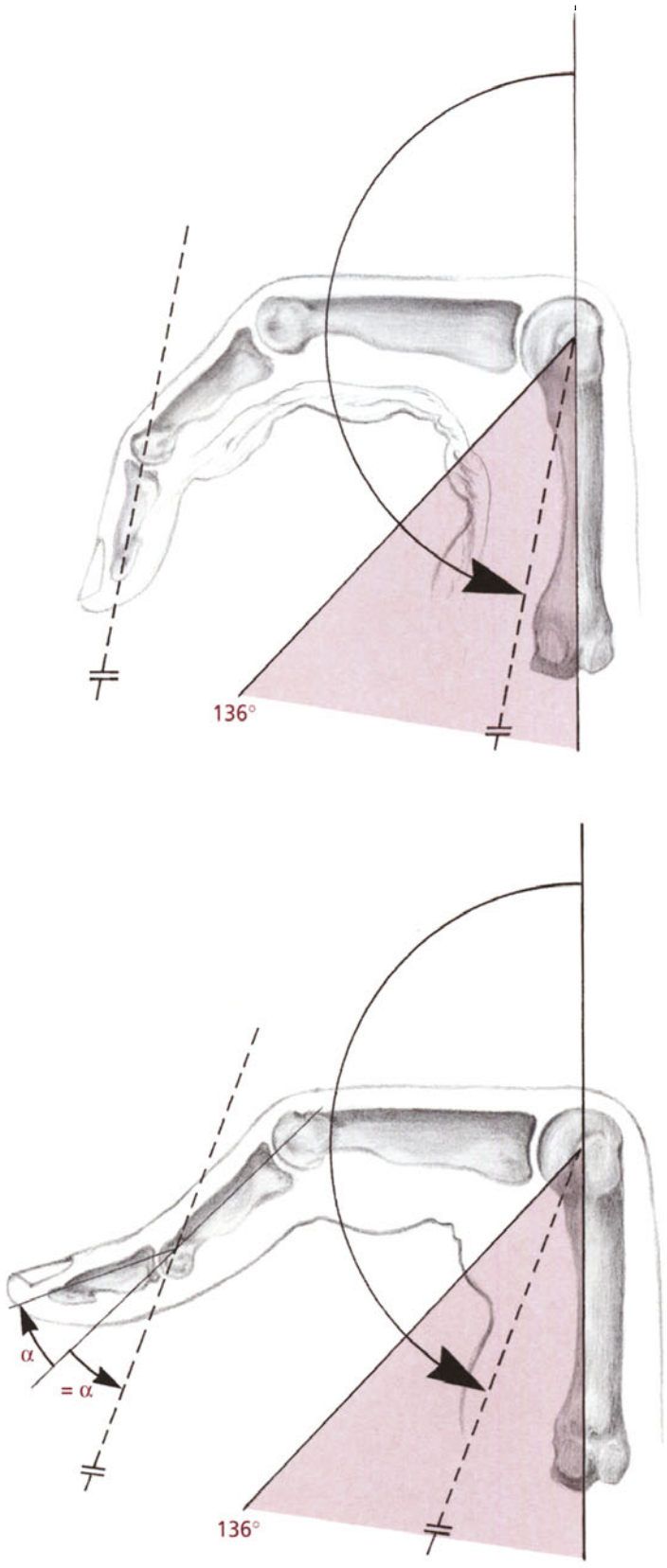


Fig. 23e. Stage 4: Flexion contracture above 135 degrees. If the distal phalanx is hyperextended, the degree of this hyperextension is added to the sum of all contractures.

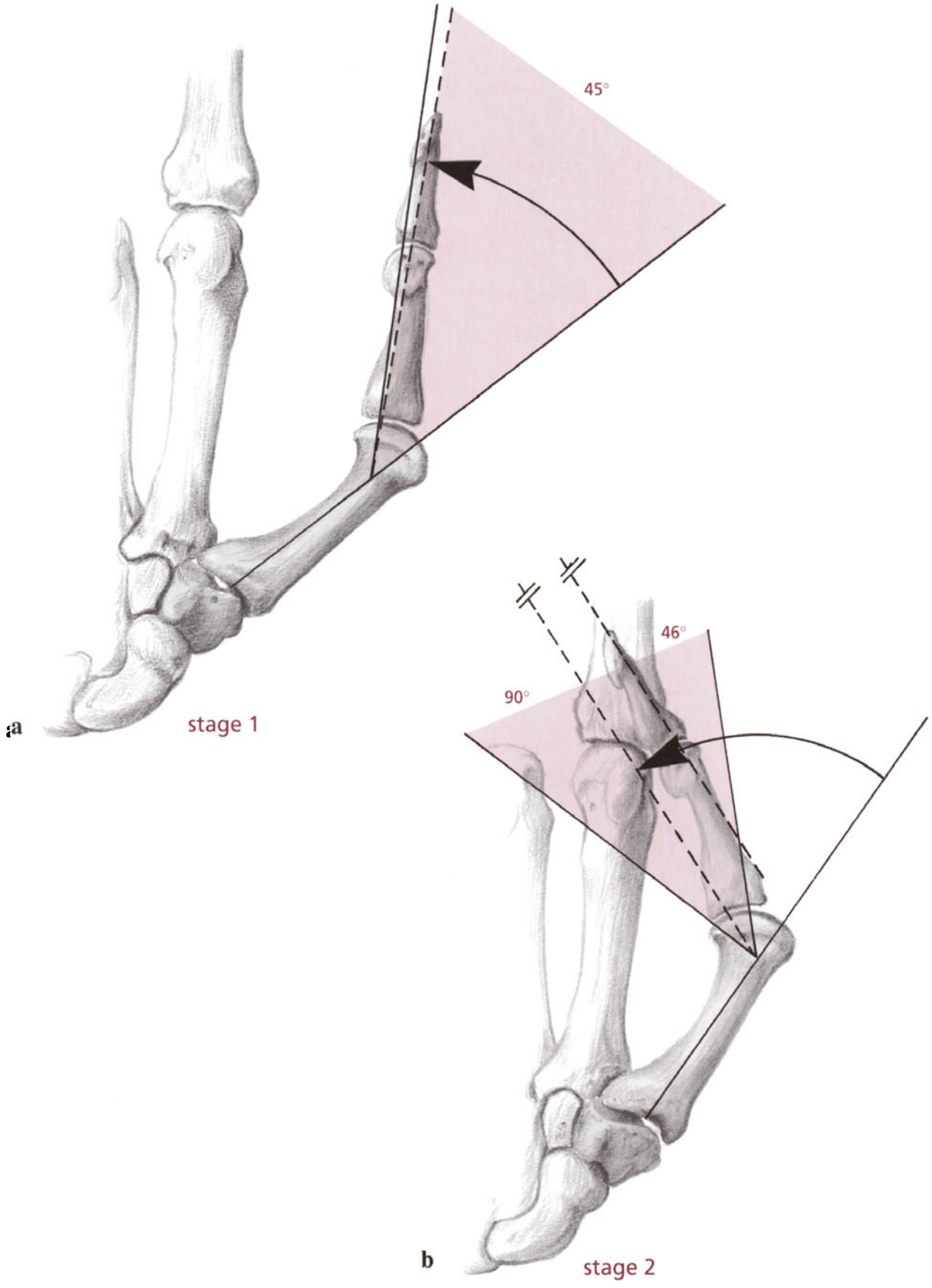
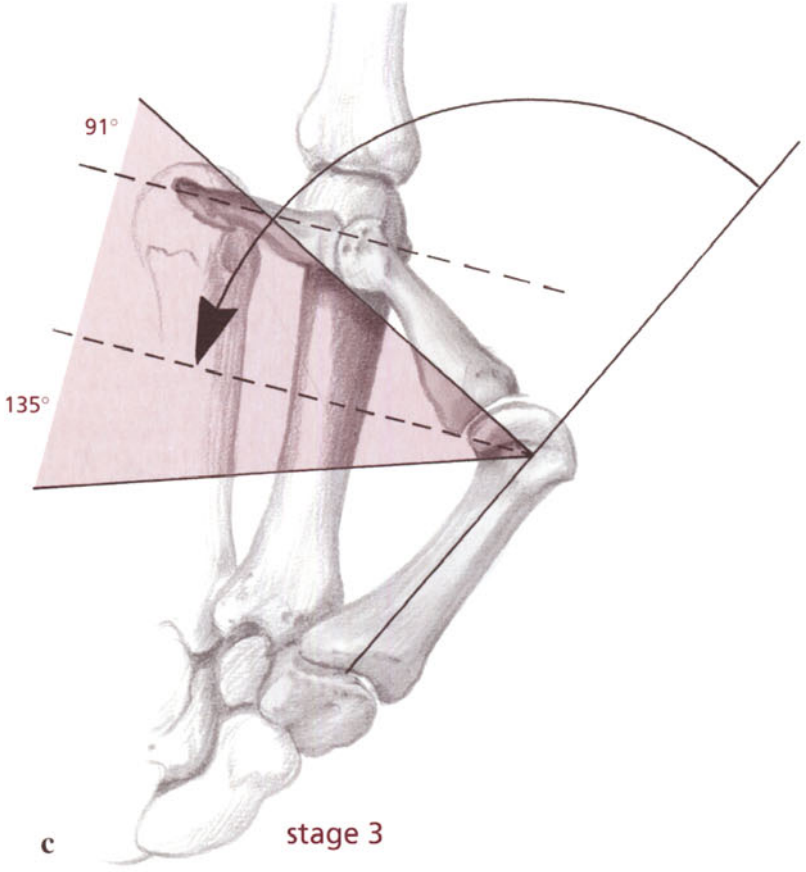
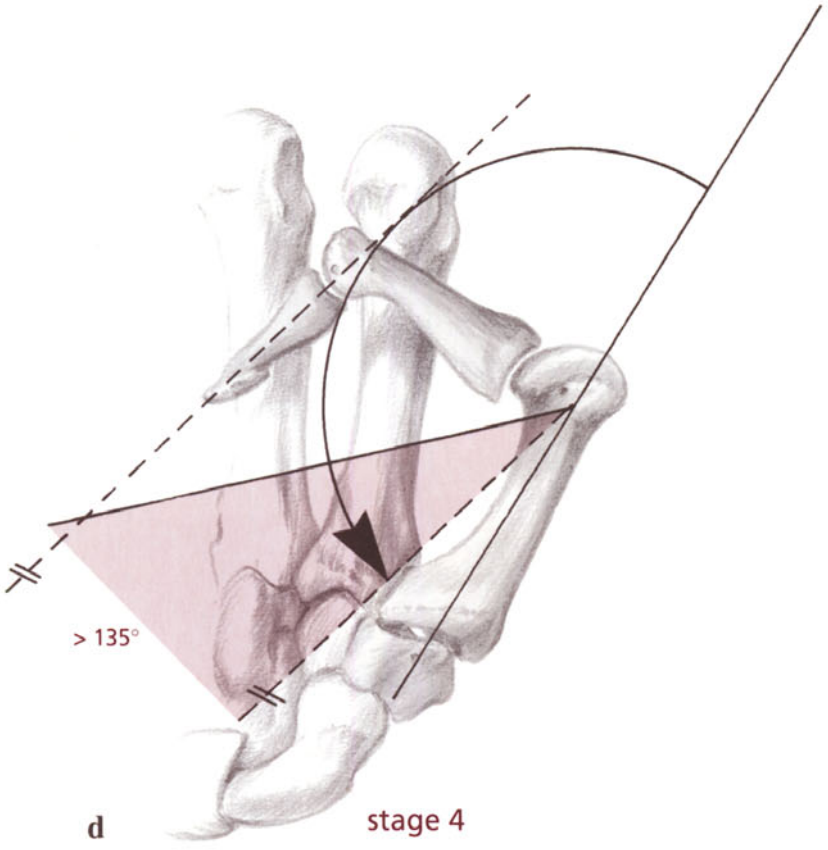


Fig. 24a–d. These 45-degree steps also apply to the thumb ray.


91°
135°
c
stage 3


> 135°
d
stage 4

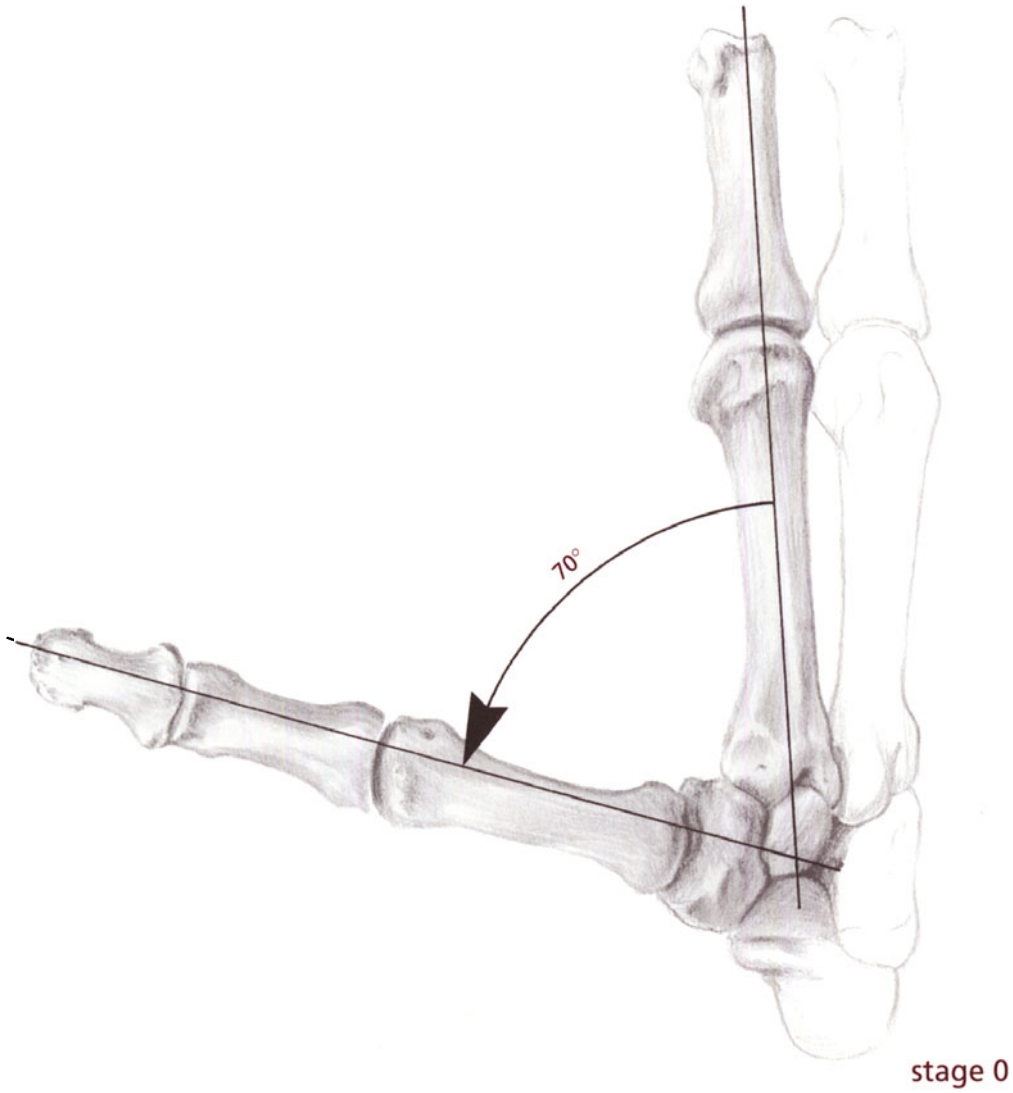


Fig. 25a. Classification of the loss of abduction of the first web space. Stage 0: neither nodule nor adduction. Stage N: nodule. Abduction to 70 degrees.

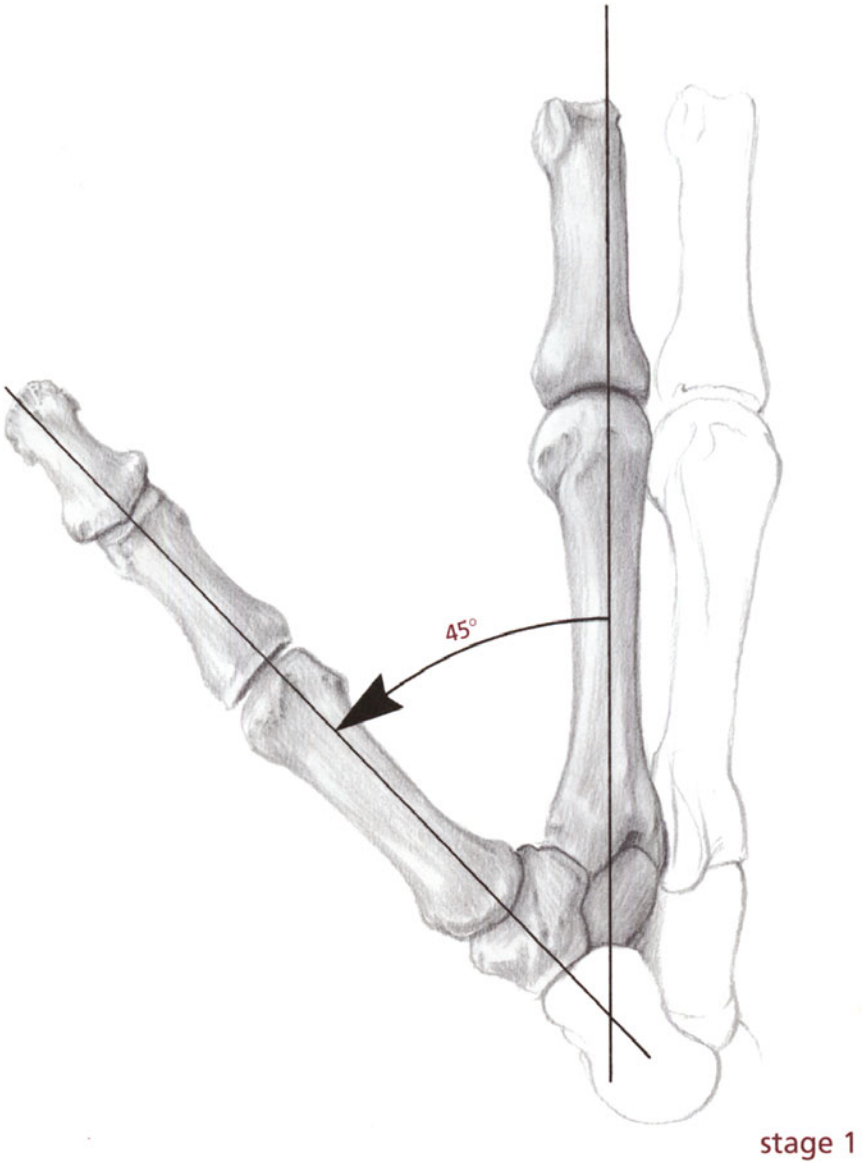


Fig. 25b. Stage 1: abduction angle between 45 and 30 degrees.

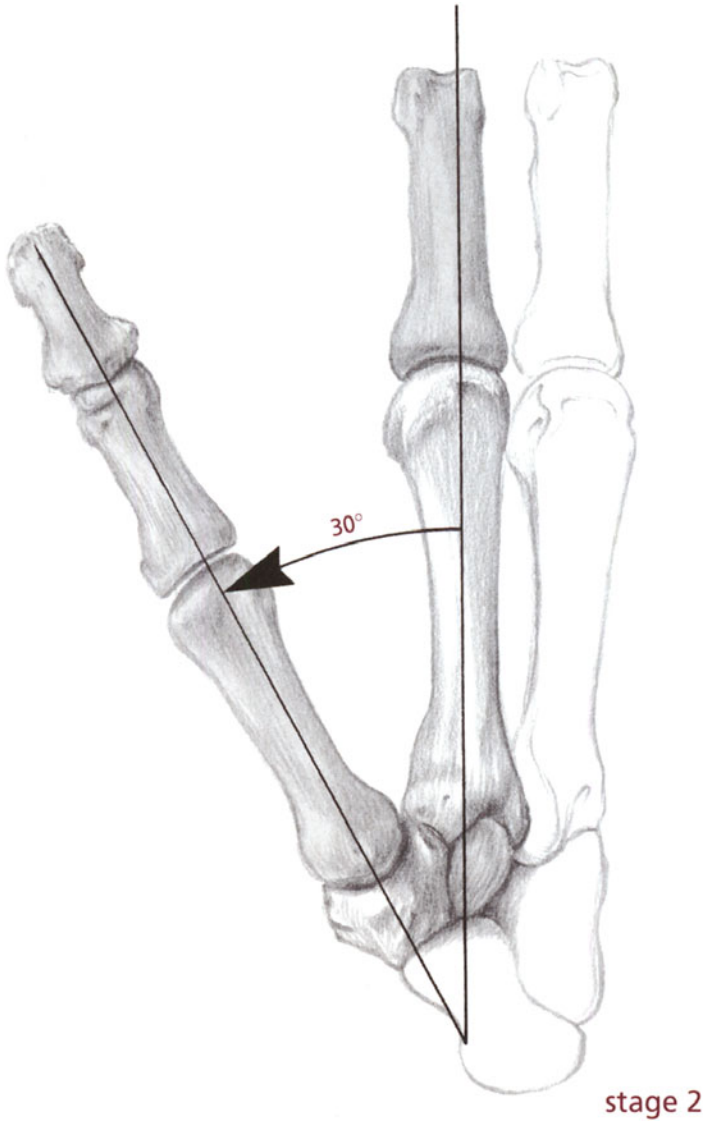


Fig. 25c. Stage 2: angle between first and second metacarpal 30 to 15 degrees.

Fig. 25d. Stage 3: adduction contracture between thumb and index < 15 degrees.

11 Conservative therapy

As the real cause of Dupuytren's disease is unknown to this day, it is not surprising that there are many different and conflicting suggestions for various type of management. Because of the multitude of suggestions, some of which are not really important to physicians, we will restrict our presentation to the discussion of radiotherapy and pharmacological treatment (Dahlke, 1992; Dahmen et al., 1967; Herbst et al., 1985; Reifferscheid, 1951; Voigt et al., 1980; Lamb, 1981; Leclercq et al., 2000).

Efforts at pharmacodynamic elimination of the pathological fascial tissue were first published by Bassot (1965, 1969). Local injections with a mixture of trypsine, hyaluronidase, and mixed with a local anesthetic, by Hueston (1971, 1974) erroneously called "enzymatic fasciotomy", only lead to short term improvements of digital contracture, according to Lamb (1981), who thinks such techniques do not present a suitable long-term treatment for Dupuytren's disease.

Badalamente and Hurst (2000) report the ineffectiveness of using collagenase derived from clostridia, in doses of 300–9,600 units. In their phase II study, only doses of 10,000 IU improved the metacarpophalangeal and proximal interphalangeal flexion contractures (28/35 metacarpophalangeal contractures showed a normal result, 2/34 had an extension deficiency of 5 degrees one to 14 days after the injection. The process normalized in four out of nine patients after treatment; one achieved an extension deficit of 10 degrees; a deficit of 15 degrees remained in two PIP patients. The collagenase treatment failed in two patients; these underwent secondary fasciectomies).

Temporary side effects such as dysesthesia, edema of the hands, and hematoma, which resolved within one to two weeks, occurred in all treated patients. The two-year recurrence rate was 25%.

Because of higher morbidity in his patients—with the same rate of relapse—McCarthy (1992) speaks out against enzymatic fasciotomy. It remains to be seen to what extent the disadvantages (tendon ruptures due to enzymolysis, neurovascular lesions, etc.) can be reduced by a more sophisticated application, e.g. under sonography control.

Antioxidants eliminating radicals are also called "scavengers". The scavenger used for intralesional application in fibromatosis, orgotein (Eckert and Koch, 1987; Talke et al., 1988) has been delisted by the health authorities.

In the context of the working hypothesis of oxygen radicals possibly inducing or triggering Dupuytren's disease, the literature repeatedly mentions the xanthine-oxydase/dehydrogenase inhibitor allopurinol (Joyce, 1987).

Murrel et al. (1987) showed a significant range of motion improvement of 7–8 degrees after administering 300 mg allopurinol for three months. There are claims of individual patients achieving complete finger extension after taking the medication for over two years.

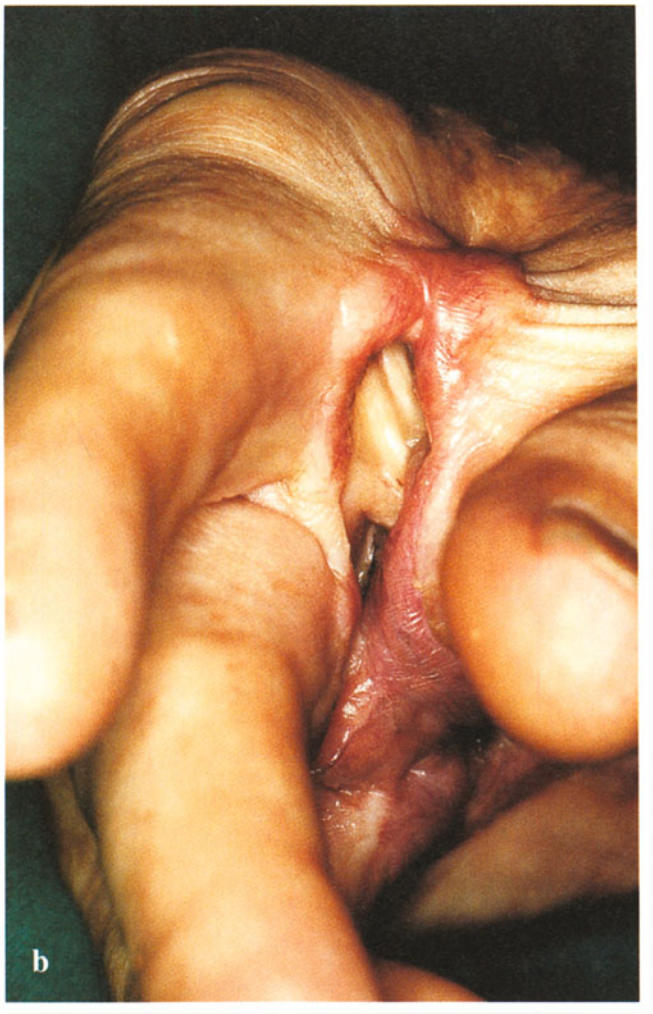
To what extent colchicine and vinblastine are capable of preventing the intracellular accumulation of fibrous long-spacing collagen (FLS) by interfering with the microtubular secretion pattern, may be the subject of future studies (Dominguez-Malagon et al., 1992).

Lappi et al. (1992) mention the basic fibroblast growth factor—saporin as another effective pharmacological option. Saporin is a mitogenic substance derived from the plant Saponaria officinalis (Fig. 26, Stirpe et al., 1983). This protein preparation, which has ribosome inactivating properties, is already successfully used in patients with AIDS, tumors, and graft versus host reactions.

Selenium, receptor blockers, and inhibitors of the interaction between cell and matrix (calcium blockers or integrins) are listed in the current liter-

Fig. 26. Saporin is a mitogen extracted from soapwort. The agent is used to treat Dupuytren' disease conservatively. Soapwort belongs to the genus Saponaria. Approximately 30 species are known in the Mediterranean and in Eurasia. Three species are native to Central Europe, including Saponaria officinalis (Common Soapwort). It is usually found on trailsides. This photograph of Saponaria officinalis was taken at the National Botanic Garden of Belgium in Meise/Flanders.

Fig. 27a, b. Long-term damage after irradiation for digitopalmar flexion contracture: actinic ulcers of first web space and hollow of the palm.



ature as possibly beneficial. Their effectiveness will be discussed later.

There are several reports that describe an allegedly positive influence of radiotherapy on Dupuytren's disease (Köhler, 1984; Herbst et al., 1985; Falter et al., 1991). These reports shockingly refer to even "early stages" of digitopalmar flexion contracture being treated (Keilholz et al., 1997). Most of these reports, however, ignore the most important aspect, the natural history of the disease, which can stagnate or go into remission even without treatment. The observation periods after radiotherapy are also too short, so that there are no reliable data about radiodermatosis and actinic ulcerations or other late sequalae. Such ulcera after bilateral radiotherapy of Dupuytren's disease are shown in Figs. 27a and 27b.

Radiotherapy of the hands is now regarded obsolete, especially for benign diseases, because it causes fibrosis with mechanical constriction and sclerosis of the gliding tissues; radiogenic, obliterative angiitis; lymphatic occlusions, actinic nerve lesions with reduction of motor strength and sensory reinnervation, skin alterations (radiodermatitis, depilation, sclerosis of the dermis, friable nails), and such earlier described lesions as painful ulcerations and secondary carcinomas. Most importantly, these complications are usually seen by hand or plastic surgeons—who report them to warn others—and not by the radiologists who caused them.

The literature on radiotherapy unfortunately does not mention anything about the high incidence of carcinomas from ionizing radiation or the significantly increased genetic risk (Finch, 1995; Evans, 2000).

The total radiation doses recommended for treating Dupuytren's disease vary between 9 and

42 Grays (corresponding to fractioned 900 to 4,200 rads; HERBST et al., 1985), despite our knowledge that national and international recommendations limit the total dose for professional workers to 5 rads per year (equivalent to 0.1 rad per week), because of the leukemia risk. In France, the maximal permissible dose to the hand is 60 rads per year (TUBIANA, 1988).

According to DUFOURMENTEL et al. (1988), the incidence of carcinoma secondary to radiodermatitis of the hand is 20 percent (n = 14/70 patients).

These facts underline why radiotherapy is contraindicated for benign hand conditions.

12 Surgical goals

All forms of fasciectomy aim to reduce the extension deficit and loss of finger abduction in both the radial and the ulnar type of Dupuytren's disease. Dysesthesias can be improved by excision of the connective tissue tumor (Berger et al., 1990). The operative procedure therefore aims to remove the macroscopically altered Dupuytren tissue, to correct the contracture of the proximal and distal interphalangeal joints, and to preserve the neurovascular bundles and the soft tissue coverage (Hurst, 1996). The risk of progression or relapse needs to be addressed as well.

Restitutio ad integrum *cannot* be expected, despite surgical intervention. Exclusive or adjuvant pharmacological treatment with indomethacin, extracts of Saponaria officinalis, hydrolytic enzymes, or other agents, as well as radiotherapy are ineffective and mostly contraindicated because of late sequelae (Köhler, 1984; Brenner, 2002).

13 Surgical indications

Prior to establishing a surgical indication, these facts need to be considered:

1. Although Dupuytren's disease is a tumor like lesion (according to the World Health Organization [WHO] definition), it is benign (ENZINGER, 1983; GLICENSTEIN et al., 1988).
2. Digitopalmar flexion contracture can reach a state of temporary or permanent still stand—without intervention and at any disease stage.
3. Daily activities are affected only if the disease reaches TUBIANA (1986) stage III (an extension deficit of more than 91 degrees)
4. In early stages of Dupuytren's disease, only subjective complaints, such as insecure grip or dysesthesia, constitute a relative indication for surgery.
5. Serious surgical complications will have to be weighed against the outlook of functional recovery after a successful fasciectomy before establishing a surgical indication. The natural history of the disease needs to be taken into account as well (MOERMANS et al., 1991).
6. It is known from the distribution of glycosaminoglycans in plasma that digitopalmar flexion contracture is a systemic connective tissue disorder, which is not amenable to local surgical intervention (BRENNER, 1994; BRENNER et al., 2001). In recognition of this fact it seems appropriate to use the term "Dupuytren's disease", corresponding to the French "maladie de Dupuytren", instead of Dupuytren's contracture.
7. HLA phenotypes, genetic mosaicism, positive family histories, results from twin studies, and DNA typing in migrating tribes lead to the conclusion that Dupuytren's disease is an idiopathic, genetic disorder. Surgical measures do not treat the underlying disease (MCGROUTHER, 1999).

We are convinced that it is best to operate during the early stage of flexion contracture. This minimizes the operative risk because the skin is in almost normal condition.

To determine the optimal time for surgical intervention, HUESTON's table top test (1976, 1984) is useful. It is positive if the total contraction of the metacarpophalangeal and proximal interphalangeal joints exceeds 30 degrees (HURST, 1996; BENSON et al., 1998).

If surgical interventions come too late, extension deficits (secondary contractures) may persist, as the proximal interphalangeal joint capsules are already contracted.

As the real etiology of the fibromatosis is unknown, a causal treatment is impossible.

The only choice is to operate in order to relieve the symptoms. The following standardized types of surgery are known:

(1) Nodulectomy, (2) palliative fasciotomy, (3) quasi-surgical needle fasciectomy (LERMUSIAUX et al., 1997), (4) limited excision = segmental aponeurectomy, (5) subtotal fasciectomy, (6) total or radical aponeurectomy, and (7) dermatofasciectomy (MILLESI, 1965; BRENNER, 1994, 2001).

T.E.C. and the open hand technique are only modifications that are being used in conjunction with one of the aforementioned forms of fasciectomy (LANZ et al., 1975; LEHMANN et al., 1977).

The various surgical procedures are not competing techniques, because each one has specific indications.

13.1 Thoughts on the indication in the nodular and cord stages

13.1.1 The nodular stage

The clinical Dupuytren nodule indicates an early digitopalmar stage. No impairment (e.g. in the ta-

ble top test) is to be expected, and surgery is therefore not indicated. Only persistent pain resulting from digital nerve compression with functional impairment of the hand represents a relative indication for premature nodulectomy (HURST, 1996).

Clinicians and basic scientist may, however, disagree on the definition of a "nodule." Clinically, a nodule is a palpable subcutaneous lump, but for pathologists the term describes a dense accumulation of cells.

The early Dupuytren's nodule is fibroblast-rich. Therefore, surgical intervention (a so-called "total biopsy" for diagnostic confirmation) should definitely not be undertaken because of the risk of exacerbation.

The only indication for total excision of a solitary nodule is suspected epitheloid fibrosarcoma, a rare tumor of the distal upper limb. Such a suspicion needs objective support from an imaging procedure (ERDMANN et al., 1995).

13.1.2 The cord stage

13.1.2.1 Surgical macropathology of the cords.

Fibrous cords are always a sign of a mature stage of Dupuytren's disease, characterized by a low number of fibroblasts. GELBERMAN (1980) names the "firm ligament-like tissue" a cord; it does not show a nodular "thickening."

In Dupuytren's disease, the cords develop by the fascial systems of the palm and fingers being "welded". The three-dimensional structure is spread longitudinally, transversally, and vertically (LEDDERHOSE, 1897). The standard technical term for the microarchitecture of the pre-existing connective tissue skeleton of the palm and fingers is that of a "digitopalmar continuum."

The formations radiating from the palm into the fingers are most important for the flexion contracture: the pretendinous cord, the central cord in the middle of the finger, and the spiral cord.

Additional well-known and surgically important cord formations are the natatory cord and fibromatous lesions inside the Grayson and Cleland ligaments. Figure 28 summarizes the best-known connective tissue bands of the palm and fingers. Each of these can be affected by Dupuytren's disease; if they are diseased, the anatomical structures are called cords.

For illustrative reasons, it makes sense to distinguish between the palmar fascia and the digitopalmar transition zone on the one hand and the digital fascia on the other.

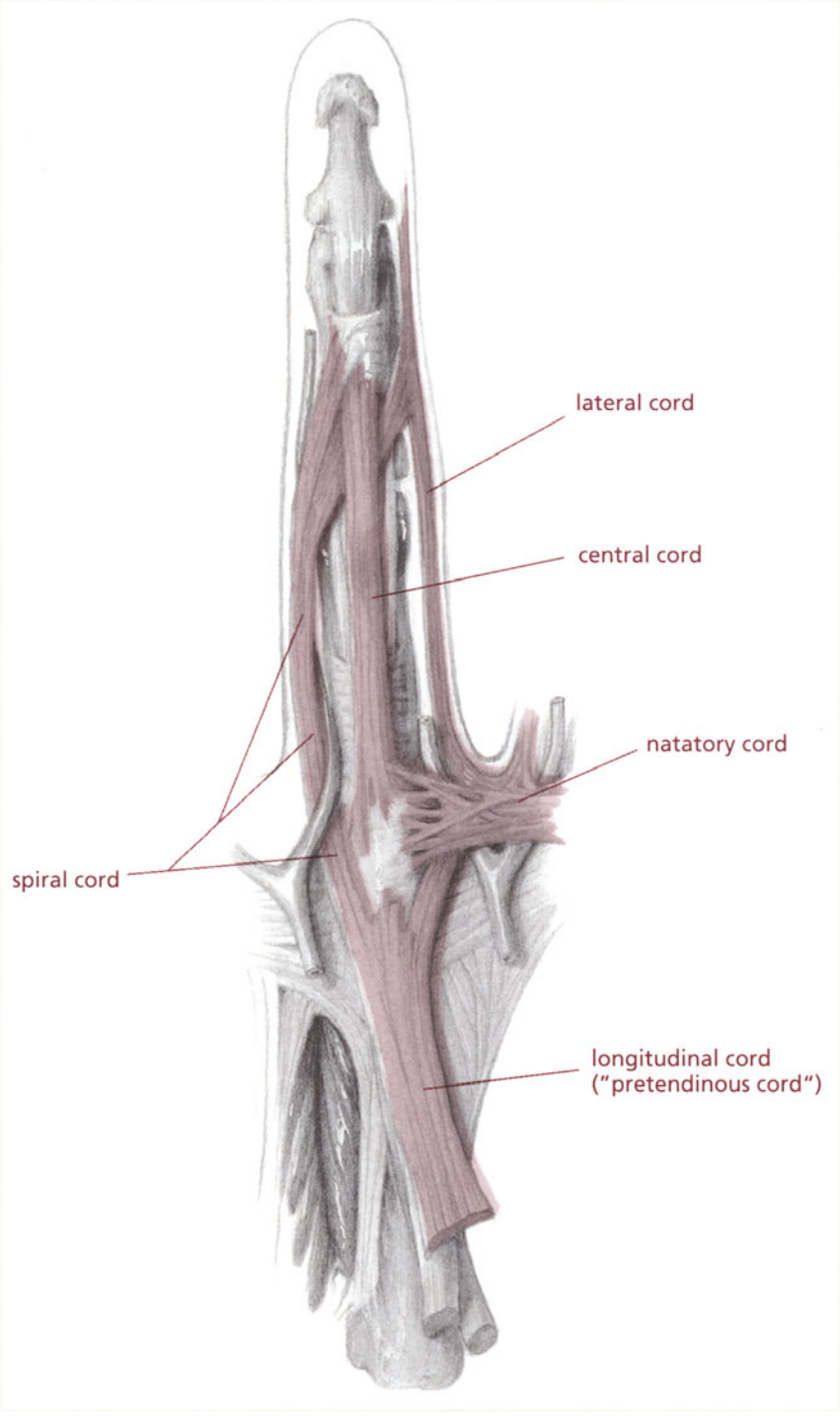


Fig. 28. Synopsis of the most important pathological Dupuytren's cords that cause contractures of the metacarpophalangeal and proximal interphalangeal joints. Perivascular cords not shown.

13.1.2.1.1 Pretendinous cord. The pretendinous palmar fascia condensates run longitudinally, overlying the flexor tendons. These fasciculi longitudinales fan out from their proximal adhesions at the aponeurosis, crossing the digitopalmar transition zone to the five finger rays. In case of Dupuytren's involvement, they become the pretendinous cords. The pretendinous cord to the thumb is less well defined.

Neither the pretendinous bands nor their diseased counterparts (fibrous cords) should be confused with the extension of the palmaris longus tendon, which may be present even if the muscle is absent. Simply stated: the central cords are peripheral extensions of the pretendinous cords.

13.1.2.1.2 Central cord. The central cord is the pathological correlate of the fasciculi longitudinales arising from the palm and located in the middle of the finger (Fig. 29). Macroscopically, a short and a long course have to be distinguished.

The shorter central cord is localized in the palm and will only occasionally lead to a metacarpophalangeal flexion contracture. In cases of a fixed contracture of the metacarpophalangeal joint, a palpable Dupuytren's nodule can often be located at the distal end of the central cord, on the palmar aspect, at the level of the metacarpophalangeal joint. In most of these cases, the skin above the palmar digital crease is adherent to the diseased nodule-cord-formation. From the short central cord, individual fibers run to the palmar digital crease, whereas others radiate into the natatory ligament.

The long form of the central cord develops as the result of simultaneous connective tissue hyperplasia with retraction of the pretendinous ligaments and the central parts of the palmar digital septa, i.e. mainly the GRAYSON ligaments. Points of origin and insertion are the palmar fascia and the proximal interphalangeal joint crease, respectively.

As a rule, the central cord does not affect the position of the neurovascular bundles, which are located on either side (MCFARLANE, 1990; MCGROUTHER, 1990).

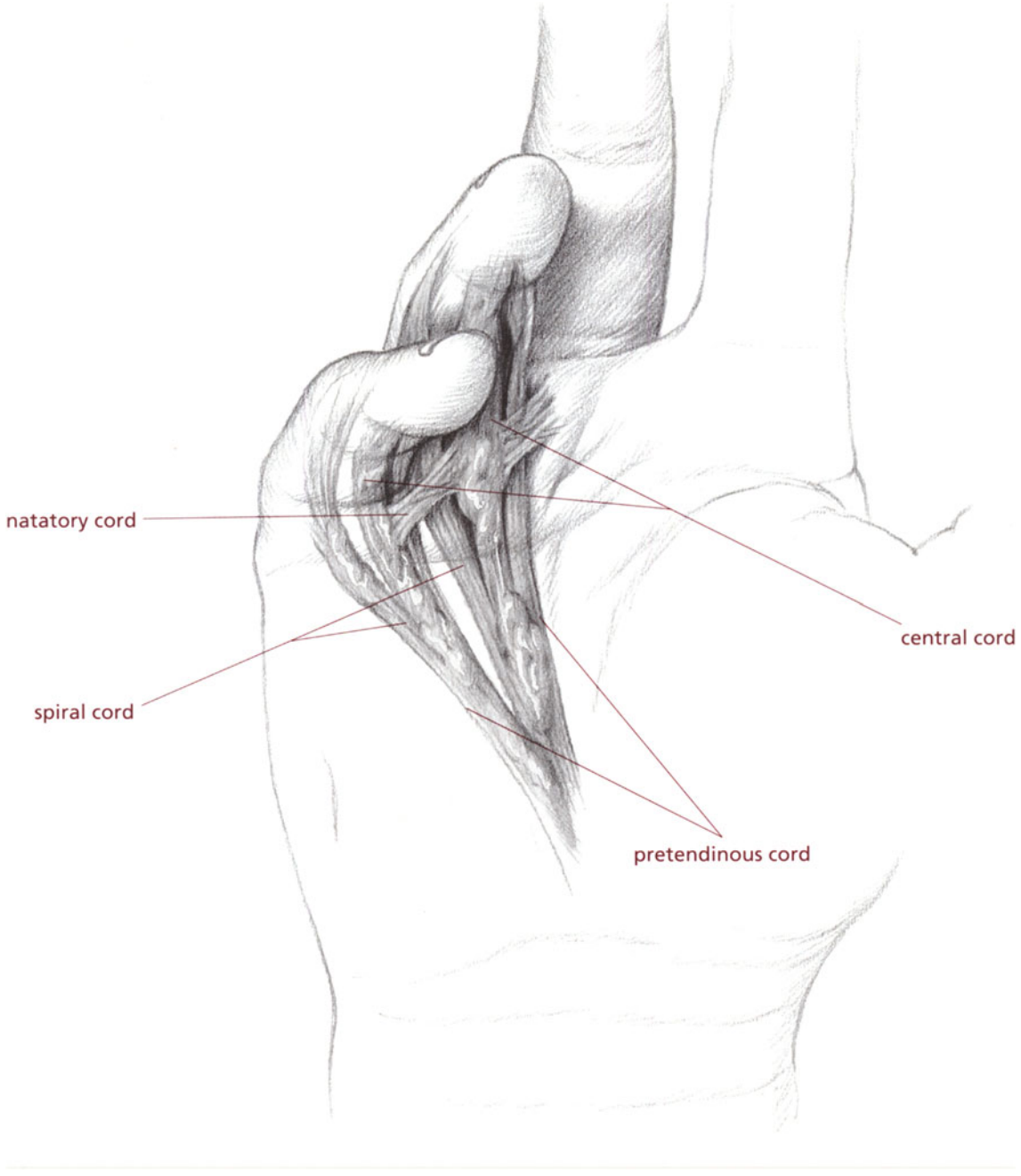


Fig. 29. Characteristic contracture of ring and little finger due to pretendinous cords, which cause flexion deformity of the metacarpophalangeal joints. Fibromatous central and spiral cords induce flexion contracture of the proximal interphalangeal joints.

13.1.2.1.3 Cords of the superficial transverse metacarpal ligament ("natatory cord"). The digitopalmar transition area has an important morphological correlate: the superficial transverse metacarpal ligament (ligamentum metacarpeum transversum superficiale). Synonymously, this ligament can be called either natatory ligament or Bourgery's transverse subcutaneous band of the natatory ligament (Bourgery et al., 1932; Zancolli et al., 1992). Some authors call it the distal transverse commissural band, just in contrast to the proximal transverse ligament of the palmar aponeurosis (fasciculi transversi aponeurosis palmaris).

When affected by fibromatosis, the parallelogram-mesh-like network of the natatory ligament, into which the vertical septa insert, causes an adduction contracture of the web space. The normally U-shaped commissure is deformed into a V-shape, which causes an adduction deficit of the fingers (Fig. 30a and 30b).

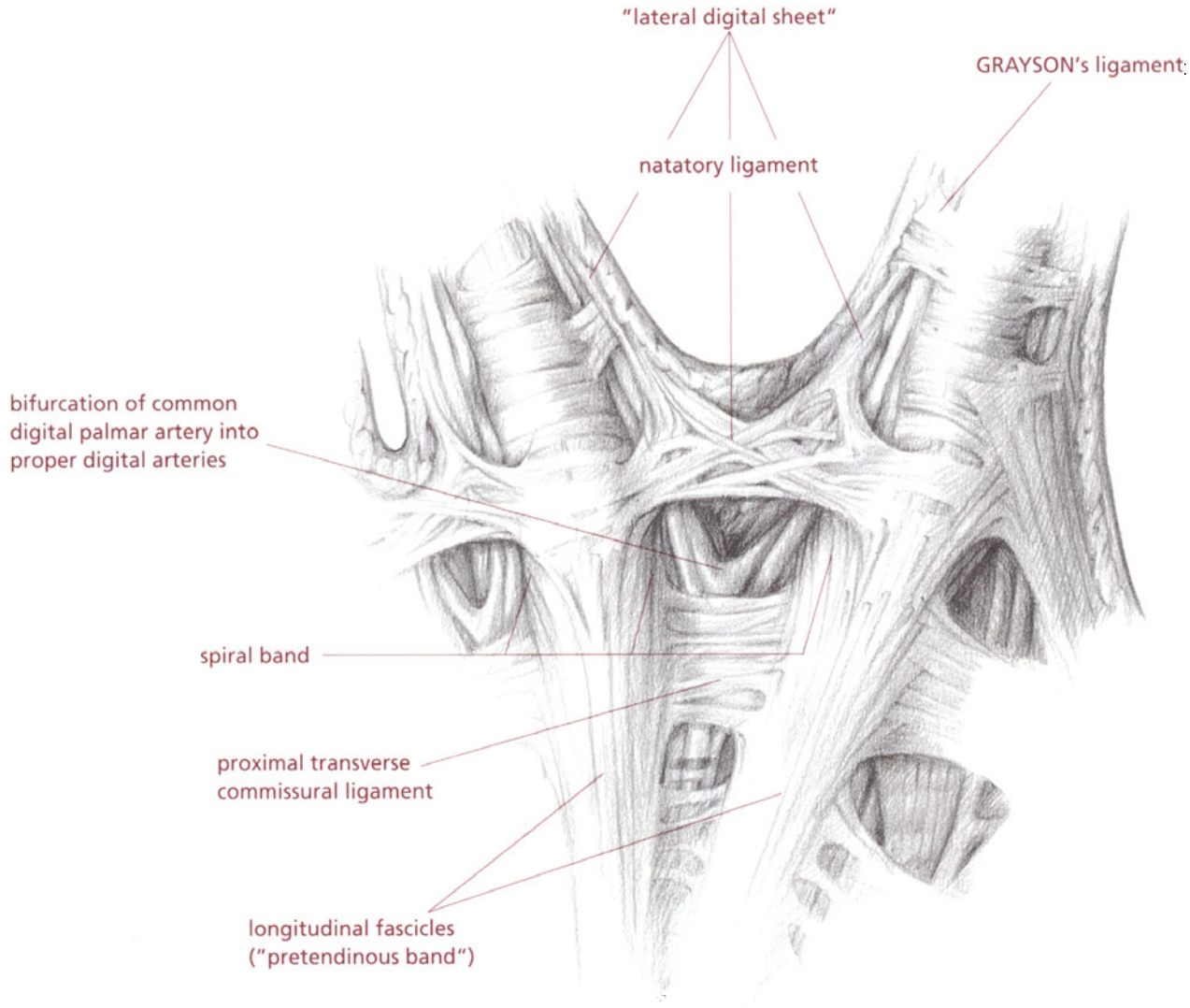


Fig. 30a. The parallelogram-mesh-like network of the natatory ligament (subcutaneous transverse ligament) is a three-dimensional chiasm of the pre-existing connective tissue fibers of the palm and the connective tissue microarchitecture of the fingers.

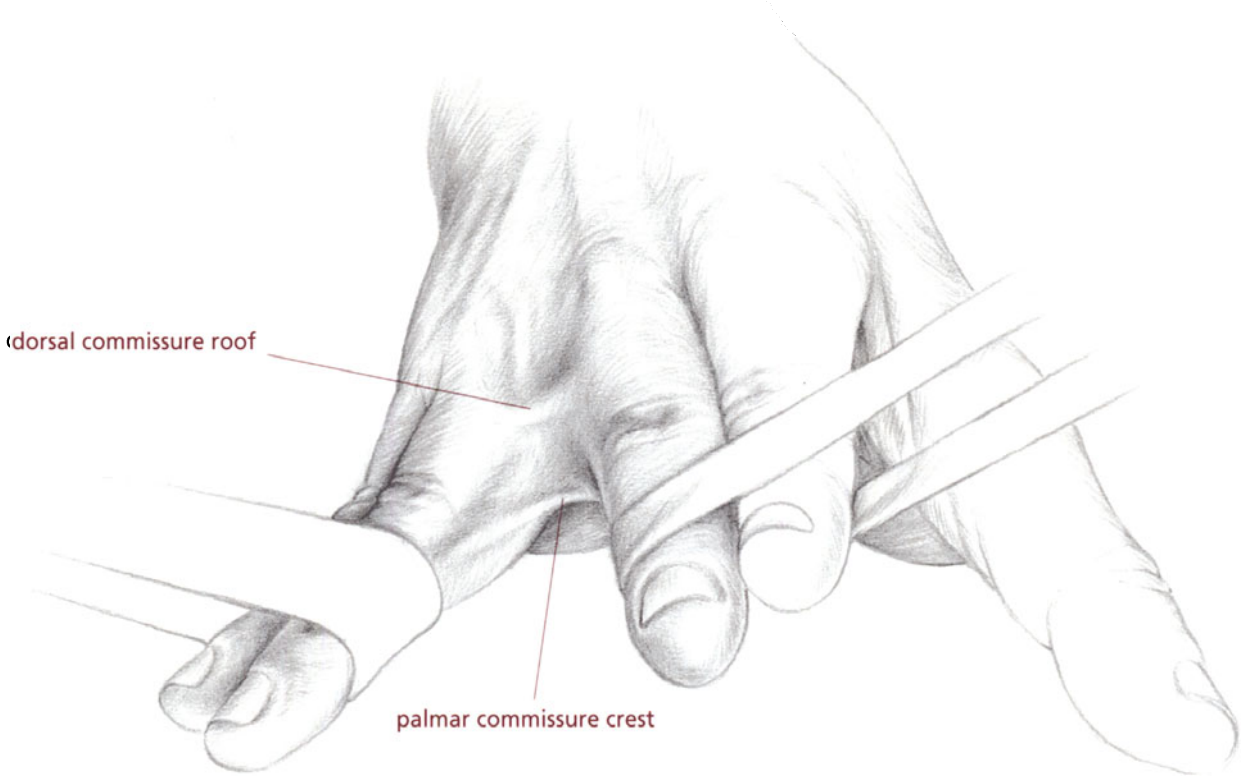


Fig. 30b. View into the web space: a palmar commissure crest and a dorsal commissure roof of the natatory ligament have to be distinguished. In case of fibromatous involvement, the originally U-shaped interdigital fold converts to a V-shaped adduction contracture.

13.1.2.1.4 Spiral cord. The spiral cord is one of the causes of the flexion contracture of the metacarpophalangeal joint in Dupuytren's disease (MILFORD, 1987). Taking off from the palmar aponeurosis, the pretendinous cord stretches into the retinaculum cutis and the connective tissue surrounding the metacarpophalangeal joint.

The spiral cord is a composite structure that develops from the pretendinous ligament of the palmar aponeurosis. It originates at the midlateral line of the metacarpophalangeal joint and predominantly inserts with its superficial portion either into the filiform skin anchoring system of the retinaculum cutis and with its deep portion either in the flexor tendon sheath of the MP joint or into the GRAYSON ligaments of the lateral digital sheath, occasionally also proximal to it (Fig. 31a, b). Its name indicates that the main fiber components of this cord are spiraling round the neurovascular bundle, displacing it medially.

Because of the varying insertions and the divergent clinical presentation, ZANCOLLI and COZZA (1992) distinguish three types of spiral cord. The type I spiral cord merges with the pretendinous cord and leads to the flexor tendon sheath of the middle phalanx. The spiral cord is most often encountered in the middle finger and occurs on the radial or ulnar side, never bilaterally. Type II has its origin at the base of the proximal phalanx and displaces the neurovascular bundle to the median line. It results from the dorsal, oblique fibers of the cribriform fascia of the dorsal digital septum and the GRAYSON ligaments fusing. Type III corresponds to type II, but originates proximally from the tendon of the intrinsic musculature. Often it continues into the abductor digiti minimi tendon.

MCGROUTHER (1999) analyzed the longitudinal course of the spiral cord, which he considers a homogenous entity, and assigns it to level 2 in his nomenclature. From the digitopalmar continuum

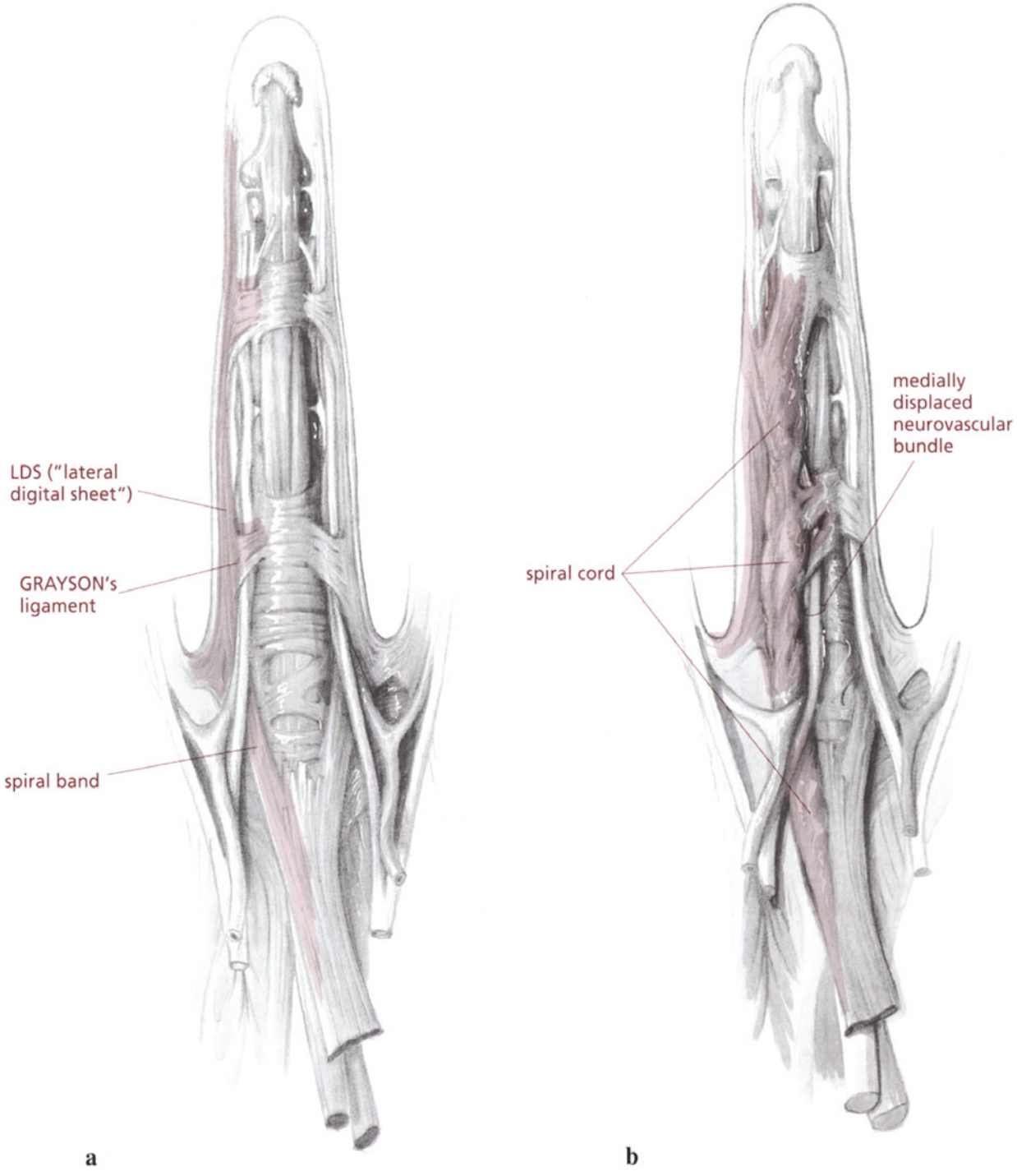


Fig. 31a, b. For simplification, only one isolated spiral cord is depicted. This cord may result in displacement of the neurovascular bundle. Left, normal condition (**a**), right, the diseased cord of a preexisting structure (**b**).

of level 2, the spiral cord extends, according to McGrouther (1999), primarily to the Grayson ligament of the middle phalanx.

Pragmatically, and important for the surgical dissection, McFarlane (1984) also distinguishes three types. Worsening flexion contracture—and the spiral cord crossing underneath—displace the neurovascular bundle—from a distal to a more superficial location, to the midline of the proximal phalanx, and finally the basis of the proximal phalanx (Fig. 32a–c).

A contracture of the proximal interphalangeal joint develops only if one of the following fascial systems is affected: the spiral, central, or lateral cord (McFarlane, 1974).

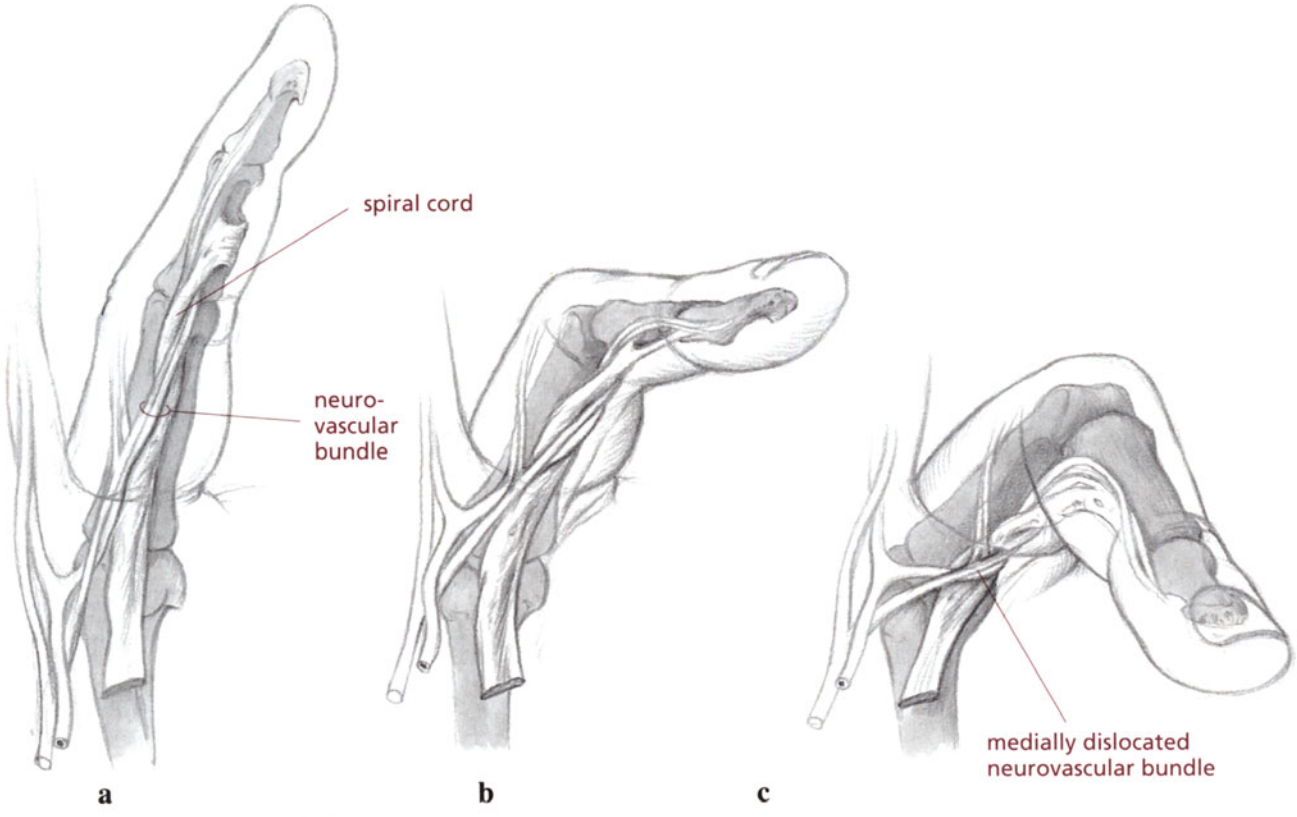


Fig. 32a–c. Spiral cord-induced flexion of the proximal interphalangeal joint increases from left to right (a–c). Increasing flexion displaces the neurovascular bundle from distal to proximal. The neurovascular bundle is eventually also displaced centrally and to a more superficial position, where it is endangered during surgical dissection.

13.1.2.1.5 Lateral cord. The lateral cord extends from the natatory ligament, which is situated on the palmar side of the neurovascular bundle, to the lateral digital aponeurotic sheet (GOSSET, 1985; [midlateral puckering zone of the superficial and deep layer of the tubular digital fascia; THOMINE, 1985]). This cord does not cause a contracture of the proximal phalanx (MCGROUTHER, 1999) (Fig. 33).

13.1.2.2 The digital fascia (digital ligaments). MILFORD (1968) distinguishes two types of digital fascia: (1) retaining of the skin ligaments and (2) retaining ligaments of the extensor mechanism. THOMINE (1985) in contrast assumes the existence of a tubular digital sheath that contains a midlateral condensate that can be allocated to anatomical ligamentous or fascial structures.

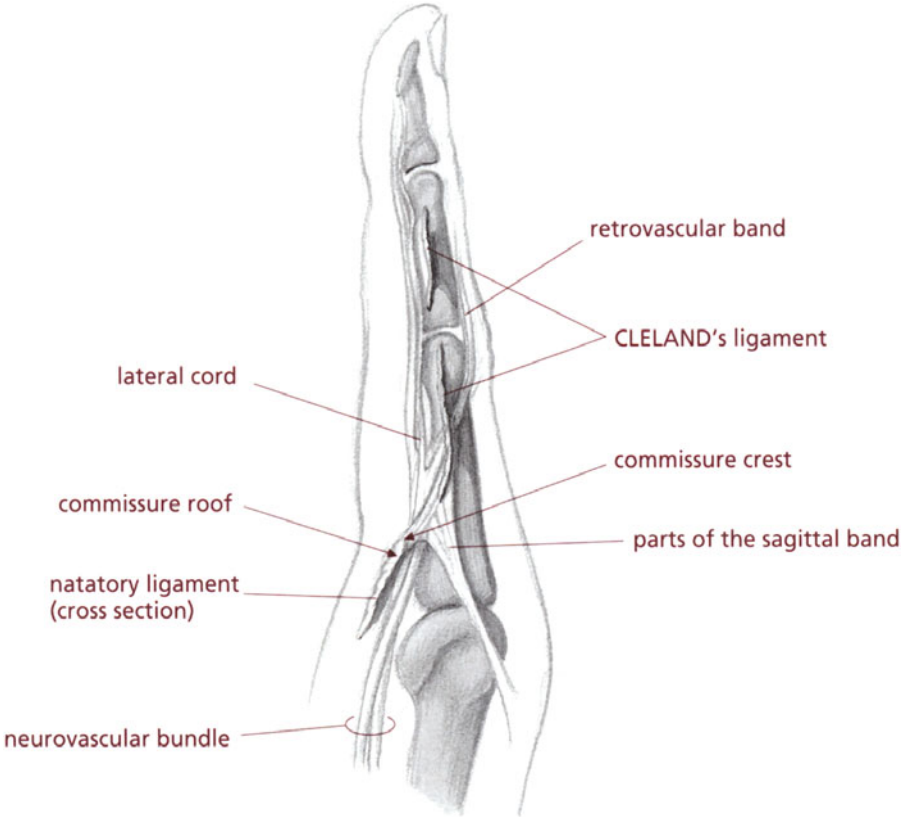


Fig. 33. The lateral cord is a centro-lateral fascial condensate of the tubular digital sheet and does not cause a contracture in itself. The palmar parts of the natatory ligament form the commissure roof and the dorsal parts jointly form the commissure crest. Both parts merge at the level of the medial phalanx and contribute to the retrovascular ligament. Because of the link between the natatory ligament and parts of the retrovascular ligament, a felxion deficit can result in individual cases.

13.1.2.2.1 The retaining ligaments of the extensor mechanism. The following structures are retaining ligaments of the extensor mechanism: the lamina triangularis (terminal joined extensor tendon), the sagittal band, the transverse retinacular ligament, and the oblique retinacular ligament (LANDSMEER, 1949, 1976; RAYAN, 1999) (Fig. 34a, b). Landsmeer's ligament extends alongside the capsule from the proximal interphalangeal joint and is rarely affected by Dupuytren's disease.

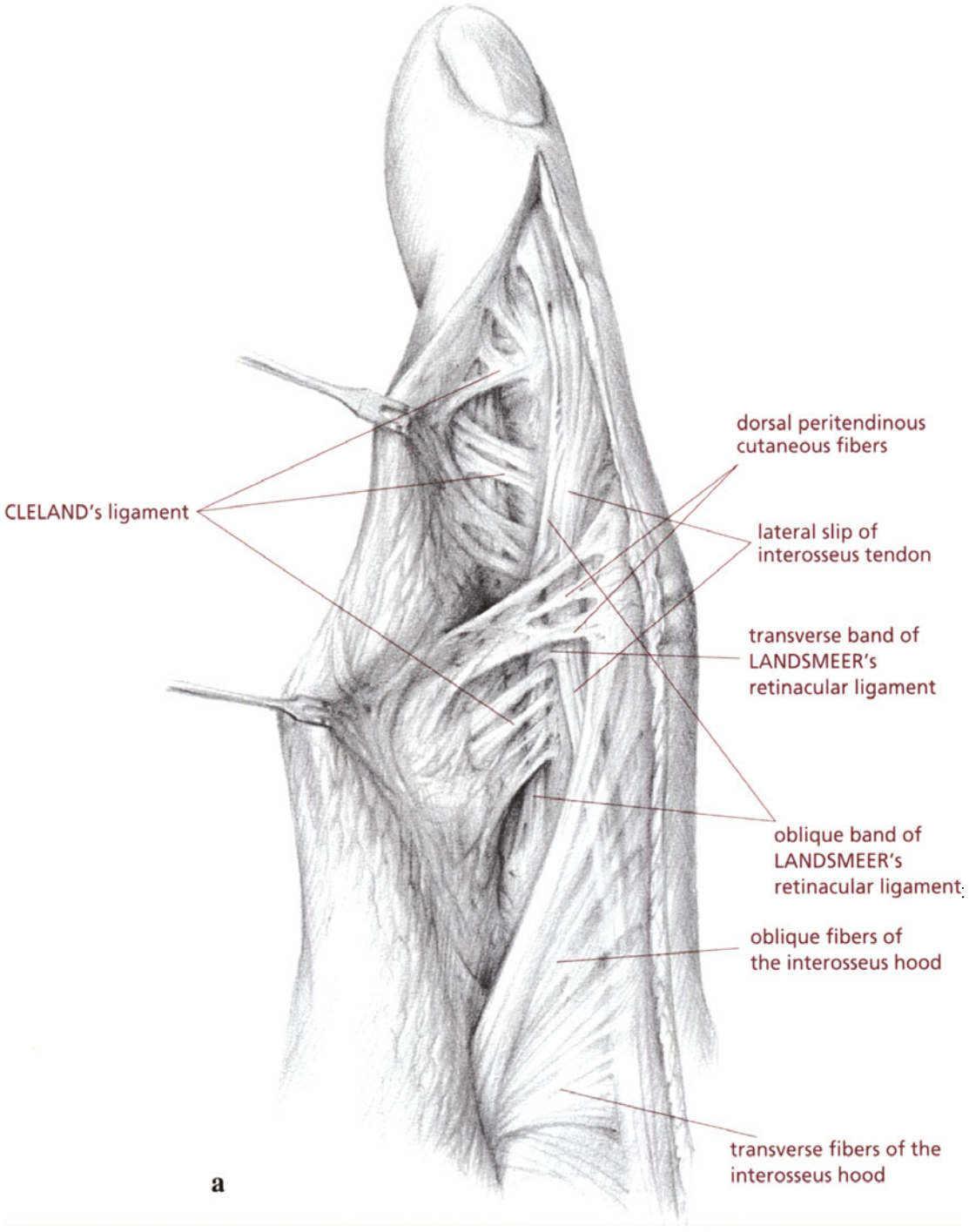
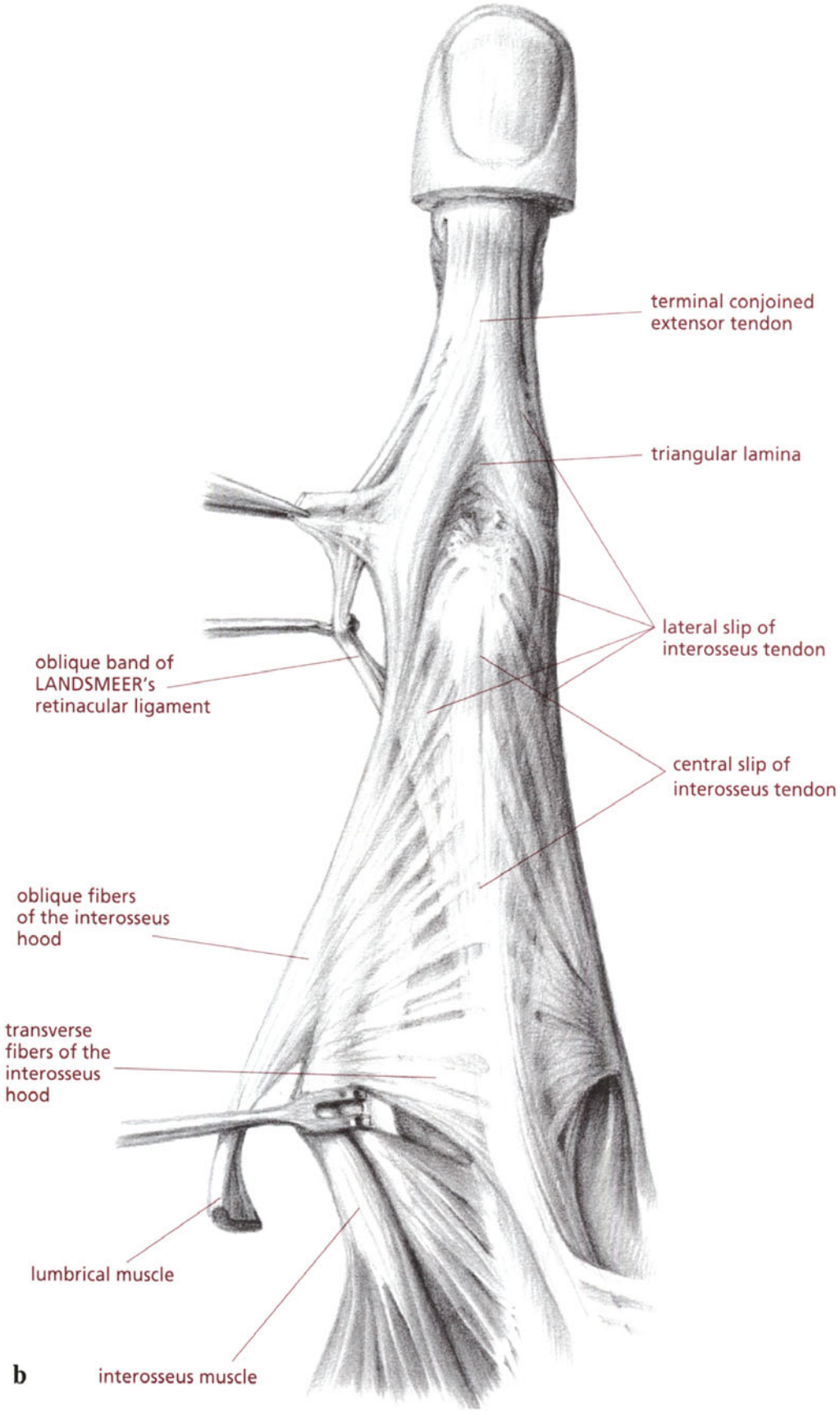

dorsal peritendinous cutaneous fibers
CLELAND's ligament
lateral slip of interosseus tendon
transverse band of LANDSMEER's retinacular ligament
oblique band of LANDSMEER's retinacular ligament
oblique fibers of the interosseus hood
transverse fibers of the interosseus hood
a

Fig. 34a, b. The retaining ligaments of the extensor mechanism: lamina triangularis (terminal joined extensor tendon), the sagittal band, oblique and transverse retinacular ligaments (Landsmeer), peritendinous fibers and finally Cleland's ligaments.


terminal conjoined extensor tendon
triangular lamina
lateral slip of interosseus tendon
oblique band of LANDSMEER's retinacular ligament
central slip of interosseus tendon
oblique fibers of the interosseus hood
transverse fibers of the interosseus hood
lumbrical muscle
b
interosseus muscle

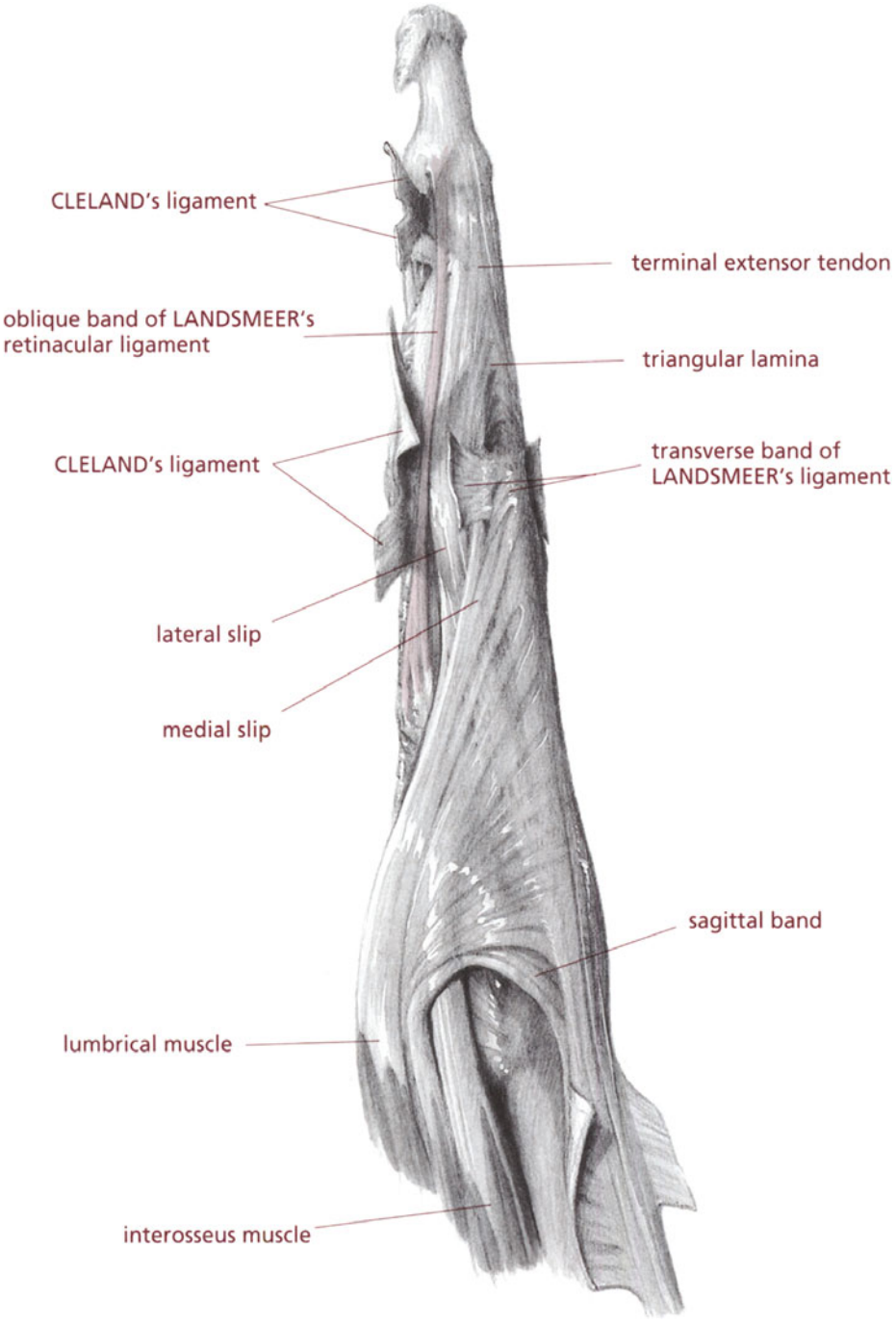


Fig. 34c. Lateral view of dorsal aponeurosis.

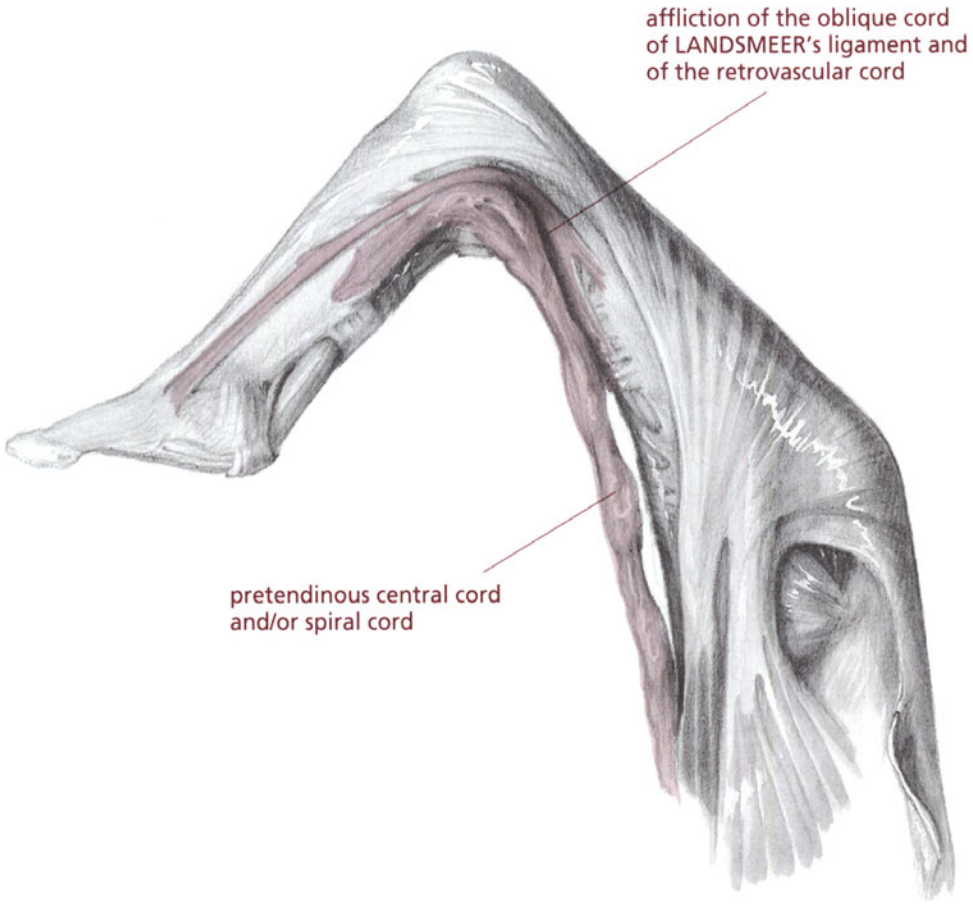


Fig. 34d. Schematic illustration depicting typical flexion contractures of MP and PIP joint and simultaneous hyperextension of DIP joint due to fibromatous affliction of the extensor apparatus (Landsmeer's ligaments).

In case of retrovascular fibrosis, however, it can cause a contracture of the proximal interphalangeal joint as well as a hyperextension of the distal interphalangeal joint. This can simultaneously affect the GRAYSON ligament. Overlooked retrovascular cords are to a certain extent responsible for recurrent contractures of the interphalangeal joints.

13.1.2.2.2 The Grayson and Cleland ligaments. Parts of the digital fascial condensates are the Grayson ligaments, which run palmar of the neurovascular bundle, and the retrovascular Cleland ligaments. At the level of each proximal interphalangeal joint, a Cleland ligament stretches in a V. These longitudinal fiber systems prevent, among others, the dislocation of the neurovascular bundle when extending or flexing the finger (Fig. 35). The occurrence of fibrosis is possible (CHRYSOPOULO et al., 2002; DUMANIAN, 2002).

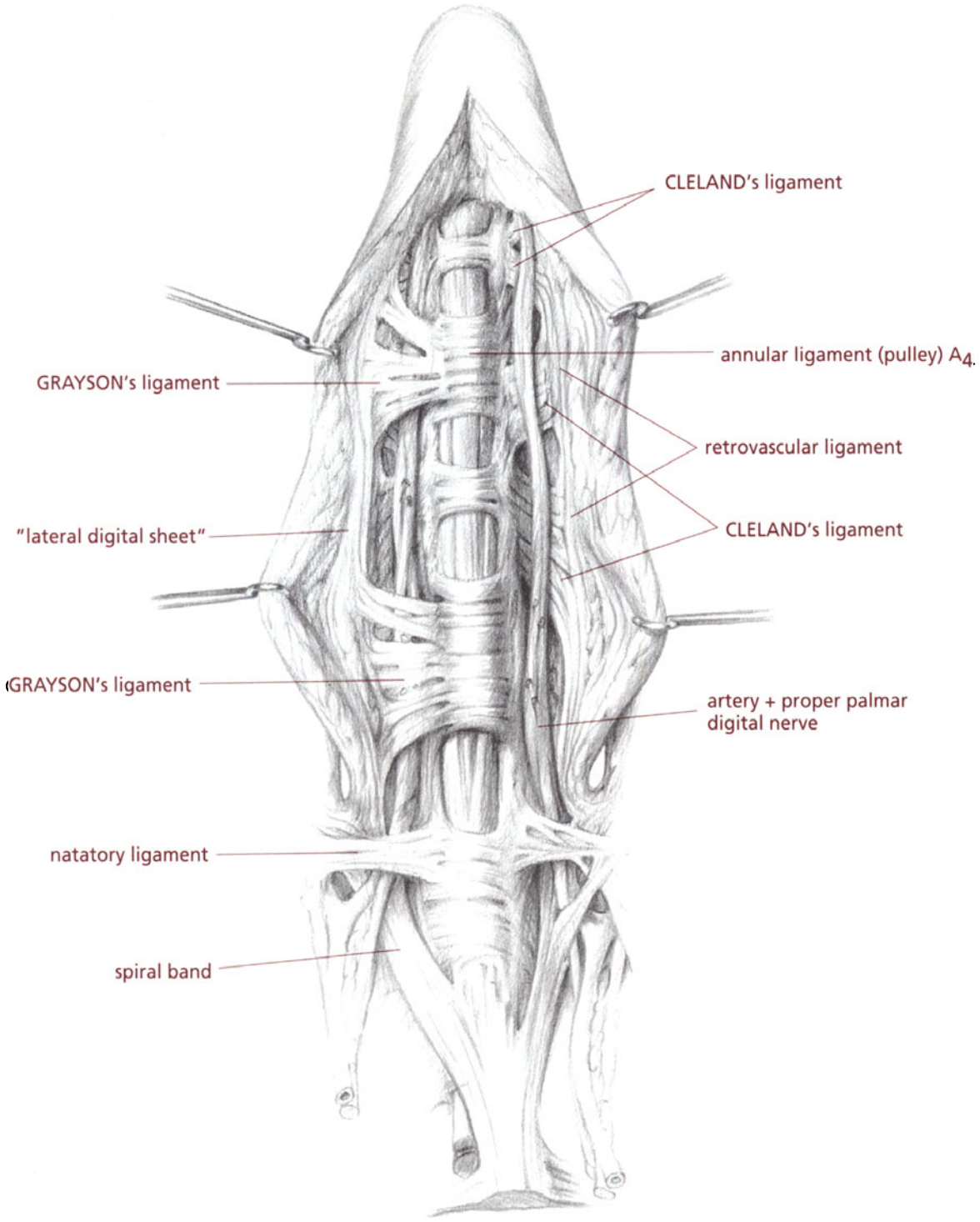


Fig. 35. The perivascular ligament systems: palmar to the neurovascular bundle, the two Grayson ligaments fan transversally. Behind the neurovascular bundles, the V-shaped Cleland ligaments extend, which are related to the retrovascular ligament.

13.1.3 Longitudinal section of the digitopalmar continuum

To simplify the palmar-dorsal topography of the various cord systems of the metacarpals and digits II to V, HOLLAND et al. (1997) and MCGROUTHER (1999) distinguish three segmental layers of the hand.

Layer 1 (superficial fiber system): Skin-anchoring system where the superficial fibers of the retinaculum cutis insert half way between the distal palmar and proximal digital skin crease.

Layer 2 (intermediate fiber system): The spiral cord runs in a corkscrew shape around the neurovascular bundle and radiates into the lateral digital aponeurotic sheet. GOSSET (1985) believes that the spiral cord runs through the digitopalmar continuum and interprets it as branching off from the palmar longitudinal fibers.

These fibers are very thin when unaffected and have been described in many modifications as retrovascular cords or lateral digital aponeurotic sheet. They insert into the natatory ligament. One part of the aforementioned fibers passes into Cleland's ligament—which is an anchoring system for the skin to the dorsal side of the proximal interphalangeal joints—and, on the other hand, into the Grayson ligaments as a ventral attachment.

Layer 3 (deep fiber system). The point of reference for distinguishing layers 3a and 3b, is the superficial transverse metacarpal ligament. The deepest part of the fasciculi longitudinales radiates proximal to the superficial transverse metacarpal ligament into the vertical septa (LEGUEU and JUVARA, 1892), in type 3a. In type 3b, the proximal longitudinal fibers run behind the transversal fibers (fasciculi transversi palmaris aponeurosis) and dorsal to the superficial transverse metacarpal ligament (ZANCOLLI et al., 1992; HOLLAND et al., 1997) (Fig. 36).

These layers of the deep fiber system (layers 3a and 3b) are of practical importance to the surgeon. SKOOG (1967, 1974) believed that the transverse fibers were never affected by fibromatosis, but a study by HOLLAND et al. (1997) showed that the deepest parts of the longitudinal fibers that run behind the transverse ligament are inevitably involved. This became obvious when HOLLAND preserved the fasciculi transversi during Skoog's selective aponeurectomy. The excision of Dupuytren tissue will therefore remain incomplete in Skoog's procedure (1974).

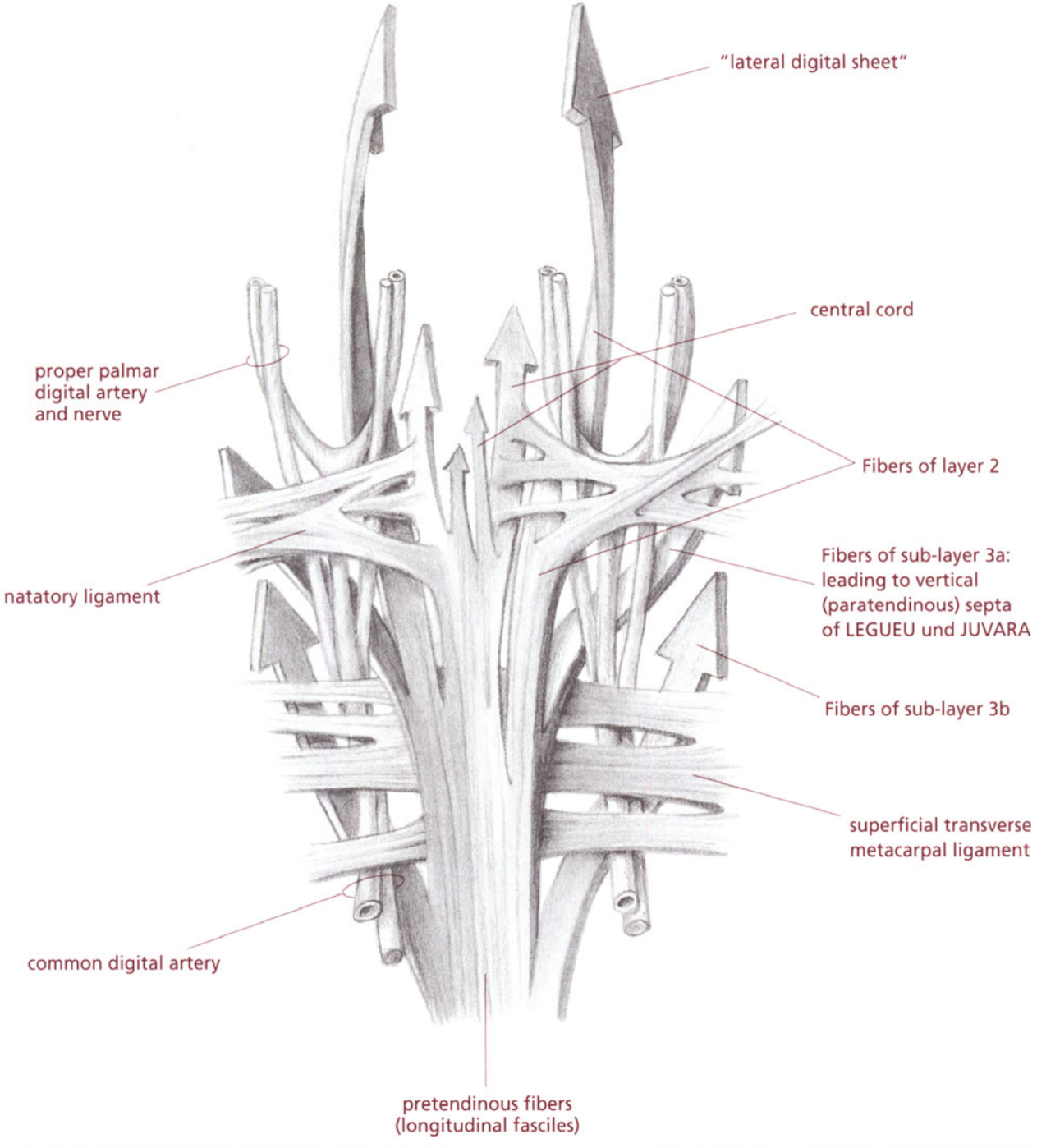


Fig. 36. The layers of the longitudinal and transverse fibers of the digitopalmar transition zone. The superficial longitudinal fibers of layer 1 insert into the retinaculum cutis. The intermediate fibers of layer 2 spiral around the neurovascular bundle. Both superficial longitudinal fiber strands pass anteriorly to the transverse fibers of the palmar aponeurosis, whereas the deeper longitudinal fibers of intermediate layer 2 extend dorsally to them. The deep fibers of layer 3 always pass anteriorly to the transverse fiber of the palmar aponeurosis, but have a different behavior with respect to the superficial transverse metacarpal ligament, which becomes the reference point. The superficial transverse metacarpal ligament divides layer 3 into two portions, namely layer 3a and 3b. The deepest part of the fasciculi longitudinales courses proximally to the superficial transverse metacarpal ligament into the vertical septa of Legueu and Juvara, this is layer 3a. In layer 3b, the proximal longitudinal fibers pass behind the transverse fibers of the palmar aponeurosis and dorsally to the superficial transverse metacarpal ligament.

13.1.4 The horizontal and three-dimensional arrangement of the digitopalmar continuum

Vertical or intermediate septa named after LEGUEU and JUVARA (1892) divide the palm longitudinally and define compartments. Each compartment consists either of a pair of flexor tendons or the lumbrical muscles with its neurovascular bundle (Fig. 37a). At metacarpal level, vertical septa are present, distal to the "Kaplan's Cardinal" line their existence cannot be demonstrated any more (Fig. 37b). For reasons of relapse prophylaxis we excise even the unaffected vertical septa.

Figure 38 summarizes the three-dimensional arrangement of the digitopalmar continuum. One can easily imagine that any fibrosis-induced adhesions will inevitably have a large impact on coordinated extension, flexion, and abduction.

Not all parts of the digitopalmar connective tissue continuum are equally affected by fibrosis from Dupuytren's disease (TUBIANA, 2000). Mainly those superficial fascial structures are affected, which stem from the fasciculi longitudinales, natatory ligament, or their digital expansions.

Because of the modified fascial architecture with the commissure ligaments taking an L-shaped course in the first web space and the unusual insertion on the thumb, the radial type of Dupuytren's contracture as well as an isolated digital cord on the ulnar border of the little finger deserve special attention. In the latter the meta-

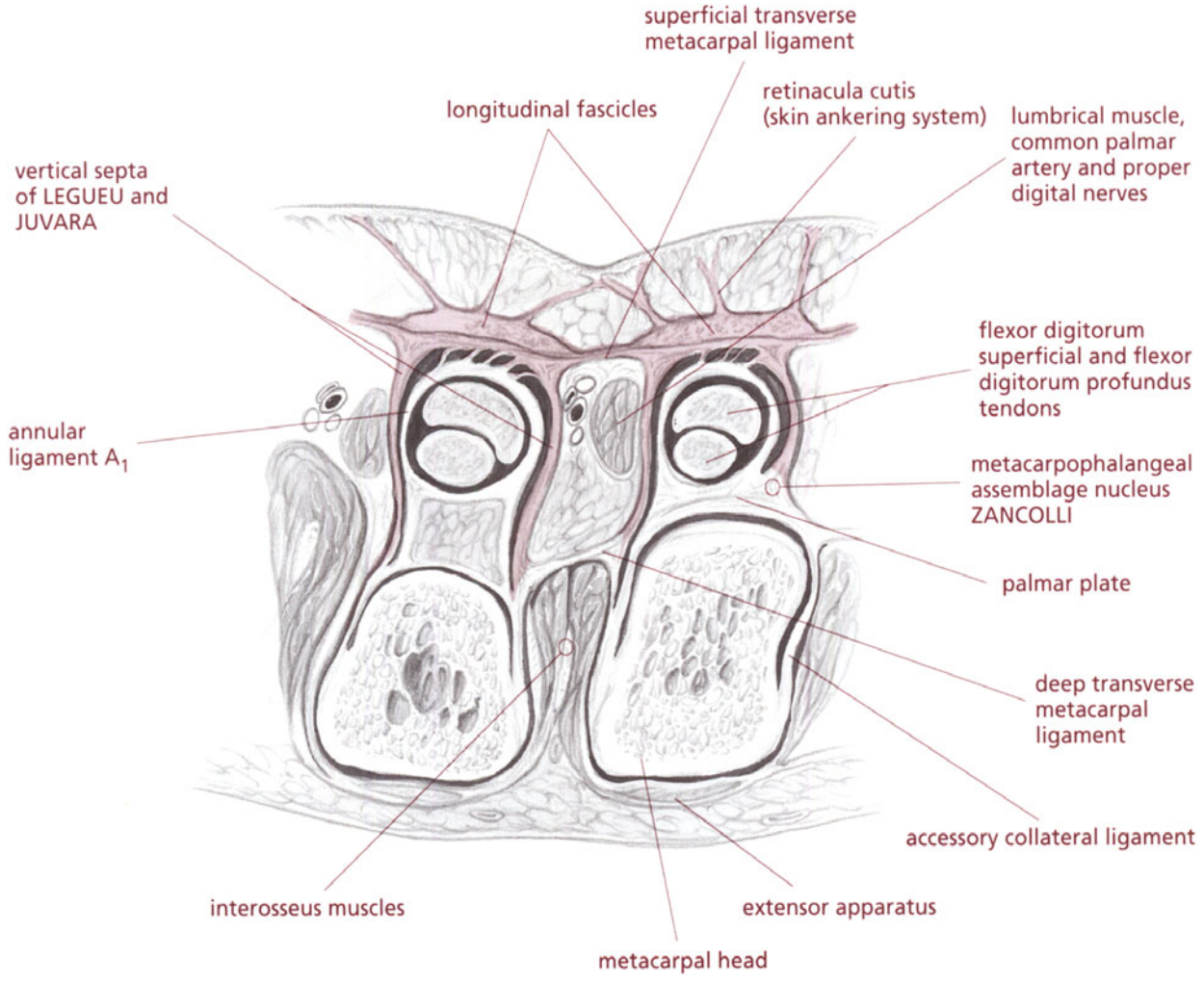

superficial transverse metacarpal ligament
longitudinal fascicles
retinacula cutis (skin ankering system)
lumbrical muscle, common palmar artery and proper digital nerves
vertical septa of LEGUEU and JUVARA
flexor digitorum superficial and flexor digitorum profundus tendons
annular ligament A1
metacarpophalangeal assemblage nucleus ZANCOLLI
palmar plate
deep transverse metacarpal ligament
accessory collateral ligament
interosseus muscles
extensor apparatus
metacarpal head

Fig. 37a. Cross section through the distal metacarpus at the level of the heads of the metacarpals to illustrate the anatomy of the vertical septa of Legueu and Juvara. The septa create two different forms of tunnel-shaped compartments. On one side, these contain the flexor tendon sheath with a pair of tendons, and on the other, one lumbrical muscle each including an accompanying neurovascular bundle.

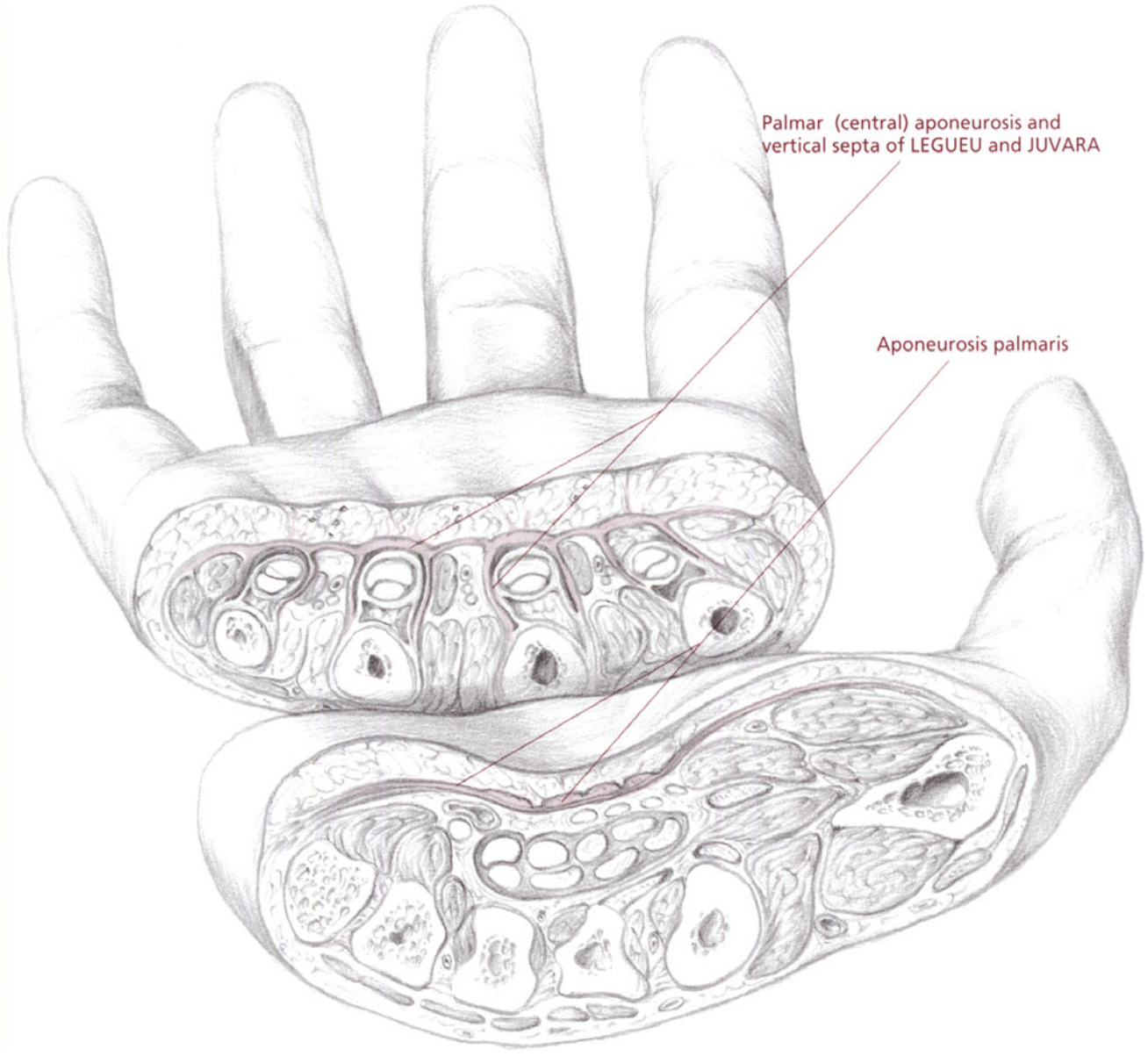


Fig. 37b. The vertical septa of Legueu and Juvara create seven compartments. Cross section of the carpus and metacarpus: the keel-shaped intermediate septa are present distally, at the level of the monticuli. Proximal to the "Kaplan's Cardinal" line the vertical septa do not exist.

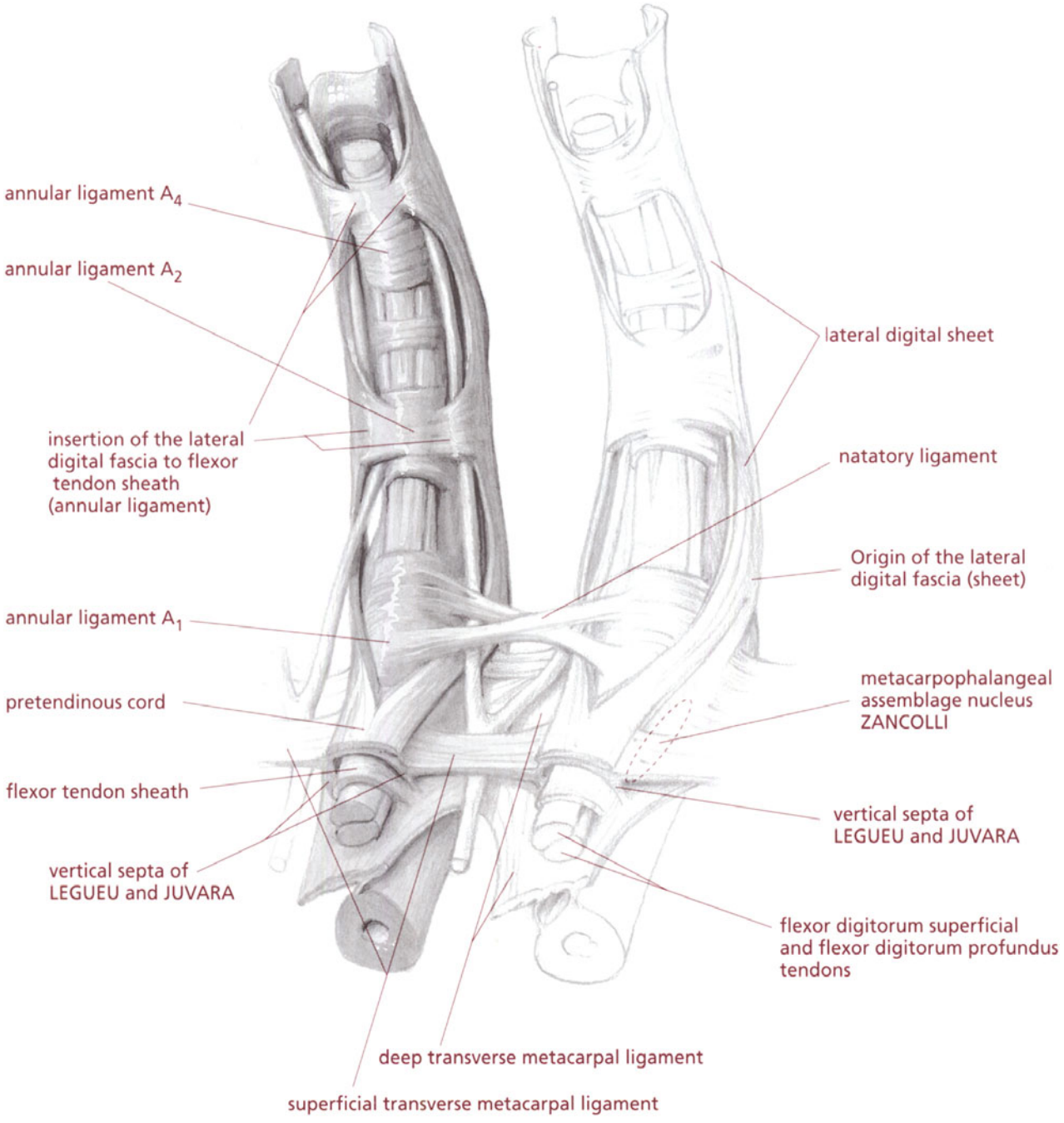

annular ligament A_4
annular ligament A_2
insertion of the lateral digital fascia to flexor tendon sheath (annular ligament)
annular ligament A_1
pretendinous cord
flexor tendon sheath
vertical septa of LEGUEU and JUVARA
lateral digital sheet
natatory ligament
Origin of the lateral digital fascia (sheet)
metacarpophalangeal assemblage nucleus ZANCOLLI
vertical septa of LEGUEU and JUVARA
flexor digitorum superficial and flexor digitorum profundus tendons
deep transverse metacarpal ligament
superficial transverse metacarpal ligament

Fig. 38. Simplified three-dimensional model of the digitopalmar continuum. Depicted is the spatial relationship between pretendinous, transverse and vertical bands (cords).

Table 1. Nomenclature and common eponyms used in the literature

Normal ligament	Affected cord
Pretendinous band (fasciculi longitudinales)	Pretendinous cord
Vertical or intermediate palmar septa (Legueu and Juvara)	Mainly unaffected but shall be removed
Longitudinal fibers extending to the finger	Central cord
Deep expansion (Gosset)	Spiral cord
Transverse fibers (fasciculi transversi aponeurosis palmaris)	Supposedly to be rarely affected in the palm (Skoog)
Natatory ligament (Grapow)	Natatory cord
Lateral digital aponeurotic sheet (Gosset)	Lateral cord
Grayson ligaments	Parts of the central, spiral, and lateral cord
Cleland ligaments + retrovascular ligament	Paricipate in retrovascular cord
Landsmeer ligaments	Part of retrovascular cord

carpophalangeal assembly nucleus is sometimes difficult to release. It receives different fibrotic contributions, including those from the ulnar end of the natatory ligament, from the pretendinous fibers of the A_1 pulley at the level of the tendinous insertion of the abductor digiti minimi muscle. Because of these remnants of the abductor digiti minimi muscle, secondary flexion contractures of the PIP joint may result, necessitating special surgical techniques (Missfelder et al., 1990).

The degree of fibrosis does not parallel the volume of physiological fascial tissue. Dupuytren's disease clearly does not simply result from hypertrophy of fascial components, but it is a genuine proliferative hyperplasia.

13.2 Mechanical strain

With regard to their mechanical attributes, Dupuytren cords have twice the tensile strength of, for example, nodules, which, on the other hand, have a notably higher stretchability (elasticity module; Afoke et al., 1998).

Millesi (1992, 1997) believes that these viscoelastic changes precede morphologic changes.

The in vitro investigations of Tarlton et al. (1998) show a linear correlation between the load applied to tissues and the release of matrix metalloproteinase-2, which is considered a marker of collagen decay.

The study of fibroblast cultures has taught us that lysophosphatic acid, serotonin, and angiotensin II make specialized fibroblasts contract. Calcium blockers, however, such as nifedipine or verapamil, and prostaglandin E_1 and E_2 block the contraction in vitro (Rayan et al., 1996; Tomasek et al., 1999).

14 Selection of surgical techniques

14.1 Indications for fasciotomy and different forms of fasciectomies

14.1.1 Fasciotomy

Fasciotomy is the simplest procedure. One needs to distinguish between subcutaneous fasciotomy, which is done by palpation only ("blind"), and locoregional fasciotomy, where the skin, subcutis, and the bowstringing cord are divided under vision. This minimal invasive procedure aims to improve hand function by simple division of the cord. It is a palliative procedure, primarily indicated in the elderly whose general condition is poor. It should be used in younger patients whenever a more invasive fasciectomy is not indicated.

The common fasciotomy can be the first step towards an incomplete or complete two-stage aponeurectomy.

A preliminary fasciotomy can also serve as a step in preparation for continuous elongation treatment (T.E.C.).

14.1.2 Needle "fasciectomy"

The "quasi-surgical," usually multistage needle fasciectomy—really a modified fasciotomy performed with a sharpened cannula—is mainly advertised in Francophone regions as a minimal invasive procedure performed under local anesthesia on patients with rheumatism or multiple associated illnesses (Badois et al., 1993; Lermusiaux et al., 1997).

14.1.3 Segmental aponeurectomy

Segmental resection, also referred to as segmental aponeurectomy, is the piece-wise resection of the Dupuytren cord, also done through an extremely small approach. Segmental aponeurectomy can be performed in patients of any age. It aims at improving the extension deficit by programmed resection of diffuse cord formations over short segments (Moermans, 1991; Andrew et al., 1991).

14.1.4 Partial fasciectomy

For primary interventions in advanced stages of Dupuytren's disease, Millesi (1981), Berger et al. (1990), and Brenner (1994, 2001) used partial fasciectomies.

Skoog (1974) referred to partial fasciectomy as "selective aponeurectomy."

Buck-Gramko et al. (1992) regard partial fasciectomy as suitable for patients of any age, for localized involvement, any degree of contracture and for recurrences.

14.1.5 Indication for the "open palm technique"

McCash (1964) returned to the basic fasciotomy techniques originally proclaimed by Dupuytren in 1832: (1) transverse incisions in the transverse palmar creases; (2) resection of just the cords causing the contracture; (3) splinting of the fingers in the extended position for four weeks after surgery; and (5) an incision that is not closed and heals by secondary intention.

The inaugurator of the open palm technique emphasized that his technique primarily leads to a release of the metacarpophalangeal contracture, but that dissection does not go beyond the proximal interphalangeal joint. A release of the proximal interphalangeal joints will always require a secondary intervention (McCash, 1964; Lubahn, 1996).

According to Allieu et al. (1986), the open palm technique is primarily indicated in multidigital Dupuytren's disease of stage III or higher, in metacarpophalangeal retraction in the very old, and also in patients in whom skin grafts or flaps should be avoided. In large studies, 18% of all patients operated upon for Dupuytren's disease

had an indication for the open palm technique (ALLIEU et al., 1986, 1988).

14.1.6 Total fasciectomy

The initial enthusiasm for "radical fasciectomy," initially falsely assumed by SKOOG (1948), SHAW et al. (1951), and MCINDOE (1958) to completely cure Dupuytren's disease, has given way to a more rational approach (OLMEDA et al., 1986).

MILLESI (1981) favors total fasciectomy in patients with good general and local conditions and in cases where the contracture of the most affected finger is less than 45 degrees.

SAKELLARIDES (1972) adds that widespread disease, as he inappropriately terms it, is an indication for total fasciectomy. Expansive fasciectomy seems to be the treatment of choice for the radial type of digitopalmar flexion contracture.

14.1.7 When to use dermatofasciectomy

Dermatofasciectomy—the removal of pathological fascial tissue with covering skin—is advised in young adults with digitopalmar fibromatosis infiltrating the skin, in cases of aggressive progression, Dupuytren diathesis, or multiple relapses (BUSCH 1882; LEXER, 1931; HUESTON, 1963; WILSON, 1997). In studies on large patient populations (n = 338), dermatofasciectomy is indicated in 13.6% of cases (BROTHERSTON et al., 1994).

14.2 Indications for additional measures

If finger extension remains limited, one needs to suspect incomplete excision of the affected digitopalmar continuum. A retrovascular cord, which lies dorsal of the neurovascular bundle—as its name implies, can in individual cases be responsible for extension deficits of mainly the proximal interphalangeal joints.

In some cases a contracture persists as a result of fibrosis affecting the flexor tendon sheath, which should be excised with the pulleys (anular ligaments and cruciform pulleys of flexor tendon sheath) left intact (MCFARLANE, 1987).

14.2.1 Arthrolysis

In addition, arthrolysis of the proximal interphalangeal joints with dissection of the check reins ligaments and possibly the accessory collateral ligaments is always indicated if more than 30 degrees of flexion persist (secondary contracture) in spite of successful fasciectomy and exclusion of all extra-articular factors (secondary shrinking of the palmar skin, adhesion or retraction of the flexor tendon sheath, shortening of the intrinsic muscles, shrinking of the palmar plate, adhesion or shortening of the reticular ligaments, scar contractures as a result of initial surgery [BELUSA et al., 1997]).

14.2.2 Arthroplasty

HAIMOVICI (1978) and MCFARLANE (1987) suggest Swanson replacement arthroplasty of the proximal interphalangeal joints in all cases where restoration of flexion and extension is not to be expected. According to HAIMOVICI (1978), alloarthroplasties provide good esthetic results and a favorable range of motion of more than 60 degrees in 73% of cases.

14.2.3 Arthrodesis

Arthrodesis of the proximal interphalangeal joints is indicated during the initial intervention, if flexion contractures exceed 90 degrees, in cases of arthritis of the proximal interphalangeal joint, or in patients who cannot comply with the postoperative rehabilitation training. It is also indicated in cases of recurrent contractures that include the proximal interphalangeal joints (PIP) (BENSON et al., 1998).

14.2.4 Amputation

In cases of flexion contractures, finger amputations are only indicated if the procedures mentioned above cannot provide a hand that is sufficiently functional to perform the daily activities, or in cases of sustained ischemia and non-reconstructible microvessels (CHUNG et al., 1996).

The fifth digit is occasionally fully contracted in spite of multiple operations; and patients request the amputation as a definitive solution (WILSON, 1979).

14.2.5 Continuous elongation technique (T.E.C.)

MESSINA's extension technique (1994) aims to straighten fingers flexed by Dupuytren's disease. It uses continuous elongation with a skeletal traction device (modified external fixateur). The external device should be installed in such a manner that it does not interfere with fasciectomy at a later date.

MESSINA (1994) sees this method indicated under the following conditions: (1) In long term digital contractures to facilitate PIP joint release; (2) if digitopalmar fibromatosis relapses; (3) if there is a lack of palmar or digital skin; (4) serious general conditions (diabetes mellitus, immune deficit, established cardiopulmonary insufficiency), (4) advanced age; (5) as a last measure to avoid amputation of a maximally contracted finger; (6) as an alternative treatment if Dupuytren's disease progresses.

T.E.C. is a time consuming procedure. After installing the external fixator it takes weeks of adjustment to achieve a favorable contracture stage more suited for a subsequent fascietomy. The inaugurator recommends his T.E.C. technique as an alternative for all those surgeons, who are not familiar with more subtle plastic surgical techniques of handling soft tissues (MESSINA and MESSINA, 1991; MESSINA, 1994).

14.3 Indications for additional simultaneous interventions

We do not combine fasciectomies or other procedures for Dupuytren's disease with a prophylactic carpal tunnel release to prevent edema, as suggested by MICHON (1977), RUDIGIER (1987) and others. The perioperative morbidity of simultaneous carpal tunnel release and fasciectomy is not significantly increased, as reported by GONZALES and WATSON ([1991]; 13% early complication rate). But NISSENBAUM and KLEINERT (1980) noted a higher rate of reflex sympathetic dystrophies (RSD) in simultaneous procedures (87% complication rate). The RSD complex includes sustained, intense burning pain sensations, swelling, and finger stiffness, as well as skin changes, disturbed vasomotor tone and an impaired sudomotor pattern (SENNWALD, 1990).

ZEMEL et al. (1987) found that in women the rate of dystrophy is 24.5% (n = 165), compared with 12.5% (n = 383) in men. Women have a higher risk for RSD. The authors therefore warn against performing simultaneous procedures in women with digitopalmar flexion contracture ("frozen hand"), whose risk is twice that of men.

BURGESS and WATSON (1987) therefore recommend to restrict the indications for a simultaneous interventions to stenosing tenosynovitis combined with an early, noncontractile, form of Dupuytren's disease. This represents one of not too many indications for a simultaneous procedure in Dupuytren's disease.

14.4 Indications in recurrent disease

14.4.1 Genuine and false relapse

Genuine relapse after surgery for Dupuytren's disease is defined as fibrous "neoplasia" in an already operated area. Disease progression or extension, however, occurs inside fascia previously not operated upon. Treatment does not change disease progression. Disease regression is always possible.

TONKIN et al. (1984, 1985) refer to false recurrences when digitopalmar contractures occur as a result of scar contracture, contractures of the joints, or muscular imbalance, primarily imbalance of the intrinsic muscles.

Surgery for genuine relapses of Dupuytren's disease is only indicated if there is an obvious functional impairment of the hand.

15 Contraindications

According to Reichmann et al. (1968), non-compliant patients and those in poor general health are not candidates for a fasciectomy. The procedure is also contraindicated in patients with progressive cerebrovascular disease and insufficiently treated chronic eczema of the hands.

Hueston (1963) considers digitopalmar contracture with strong Dupuytren's diathesis a contraindication. This disease subtype occurs in young people with a positive family history. Usually both hands are affected and ectopic connective tissue deposits are present, such as knuckle pads overlying the dorsal proximal interphalangeal joints, or Ledderhose's or Peyronie's disease (penile fibromatosis).

Lubahn (1996) considers the acquired immunodeficiency syndrome (AIDS) in addition to tuberculosis and excessive alcohol consumption (because lower compliance may be expected) relative contraindications.

Further contraindications are Parkinson's disease and strokes with residuals (McGrouther, 1999), if it is not guaranteed that rehabilitation will be successful.

Unrealistic patient expectations should be a warning alert for those active in hand surgery.

16 Informed consent

For medico-legal reasons, patients need comprehensive preoperative information.

Patients need to develop a fundamental understanding of the disease process, need to learn about the recommended and typical types of treatment including possible complications, the principles of postoperative management and the expected recovery. For legal reasons, possible treatment alternatives need to be mentioned as well, even if they are considered ineffective (STANKOVIC, 1997; HURST, 2000).

As Dupuytren's disease is treated with elective surgery, by law the patient has to be given a period of at least 24 hours before signing the consent form.

Talking about complications after they have occurred always seems to somehow imply an apology. The affected patient will then get the impression that the surgeon wants to gloss over a complication that is typical for the procedure.

The following fasciectomy complications need to be documented: persistent extension deficit, residual contractures, neurovascular lesions, soft tissue or flap necroses, graft failure, infections, "extension of the planned operation" because of unexpected intraoperative findings, loss of digits, unsatisfactory functional or poor esthetic result.

17 Preoperative work up

Detailed documentation of the loco regional status is the essential minimum; this includes measurement of joint movement for all digital joints as per the neutral-zero-method (MP, PIP, and DIP joints), finger spread (distance from the tip of the thumb to the little finger), finger extension deficit (distance from the plane of the dorsum of the hand to the free border of the nail), finger flexion deficit in centimeters (from the plane of the nail to the distal palmar crease), a basic neurological assessment, and data about the peripheral perfusion.

An obligatory measure prior to any operation is radiography in two projections (dorso-palmar neutral projection: beam from extension side to palmar side. Hand resting on foam rubber cushion at shoulder height, with elbow bent. Writer's position: oblique hand view in 45 degrees' pronation) to rule out a primary joint process as the cause of the flexion contracture. This radiograph will document any possible articular changes, which would not be adequately addressed by an operation limited to the soft tissues, or which would leave a functional impairment after fasciectomy. All this can then be explained to the patient prior to the intervention.

Extensive photographic documentation, including functional standard hand views, is also strongly recommended.

In redo operations, the clinical examination of digital perfusion should be supplemented by Doppler sonography. All cases beyond the first reintervention should undergo angiography.

The nails will have to be cleaned and trimmed before the operation. No shaving is required, and no antibiotic prophylaxis is given.

18 Instruments

An elastic Esmarch rubber bandage is essential to drain the vascular system. A tourniquet is used to maintain a bloodless surgical field.

For the operation a set of basic hand surgery instruments including a lead plate (hand plate or hand restrainer) plus bipolar cautery will suffice. Microsurgical instruments need to be available for emergency use. Surgical loupes (x 3.5 magnification) and, in individual cases, an operating microscope can be used.

19 Anesthesia and positioning

DUPYTREN (1831; 1832) presumably used alcohol and opium as anesthetics, a common practice in his day. As he did not use a tourniquet, the patient's hand was raised above heart level, and the surgeon stood behind the patient. Nowadays, we use brachial plexus block, a tourniquet, and the patient's upper extremity is positioned on a special arm table. The surgeon sits comfortably next to the arm table and uses surgical loupes (MCGROUTHER, 1988; WYLOCK, 1997). The arm is kept in about 90-degree abduction. The tourniquet is inflated to 300 mm Hg, in hypertensive patients it is inflated to 100 mm Hg above the systolic blood pressure.

An alternative to the usual forms of anesthesia, such as mask or endotracheal anesthesia, is BIER's intravenous regional anesthesia of the arm (IVRA, "Bier block"). Claims of this technique being associated with excessive and early post-operative incisional pain are unjustified. Premature cuff deflation is a known risk. IVRA is for example contraindicated in diabetic patients (SCHUHL et al., 1988; GOERIG et al., 1999).

As an exception, minimal invasive procedures such as fasciotomies or segmental aponeurectomies (MOERMANS, 1996) can be performed under local infiltration anesthesia. For adequate visualization, a tourniquet is obligatory. Without general anesthesia, the tourniquet can be tolerated for up to 20 minutes.

20 Operative technique

20.1 Remarks on skin incisions

Individual topographic circumstances need to be considered when planning skin incisions. Dissected flap require an adequate vascular supply. Surgical access has to meet several technical requirements: (1) sufficient exposure of the neurovascular bundles to trace visualized digitopalmar cords and Dupuytren nodules if needed; (2) minimal subcutaneous undermining of flaps; (3) minimal risk of wound complications; (4) optimal conditions for early postoperative mobilization; (5) prevention of scar contractures; and (6) provision for local wound margin and flap advancement or transposition.

Longitudinal incisions were shown to be superior to transverse incisions because they require less undermining and result in lower perioperative morbidity.

20.2 Approaches to fingers and palm

Figure 39a, b summarizes the approaches used for minimal invasive fasciectomies.

For partial fasciectomies, we prefer to place a midaxial longitudinal incision with serial Z-plasties (Iselin et al., 1951) over the palm and most severely affected fingers, or the zigzag longitudinal Bruner (1954, 1967) incision. For total fasciectomies, however, we favor Millesi's (1981) Y-shaped access in the center of the palm.

The most often used longitudinal incisions on the fingers have several advantages: (1) good intraoperative visualization of the affected Dupuytren tissue and the intact, primarily longitudinal anatomical structures; (2) the incision can be extended any time; and (3) longitudinal incisions permit a tension-free wound closure (Buck-Grammko, 1969; Tubiana et al., 1990). Longitudinal incisions allow for a speedy dissection and minimize tissue trauma. In summary: incisions should work for the surgeon; the surgeon should not work for the incision (Peacock, 1976).

If the finger is affected by only moderate flexion contracture, Bruner incisions will be the favored surgical method, whereas Z-plasties permit maximal lengthening in longitudinal incisions.

Bruner's zigzag incisions are made from the palmar digital crease to the opposite interphalangeal joint crease (staying proximal to distal interphalangeal joint crease) of the same finger. They run at 90-degree angles and do not cross the midlateral line. Some limited length can be gained by using V-Y flap-advancement, which will be discussed later (Fig. 39c and Fig. 49a–e).

It may occasionally be necessary to connect adjacent digitopalmar longitudinal incisions by additional palmar transverse incisions. This is the principle underlying Skoog's technique (Skoog, 1985; Tubiana et al., 1992). Combining the incisions to create a single Y-shaped incision through "Kaplan's Cardinal" line (horizontal line between first web space and the hook of hamate) is an alternative in ulnar cases of Dupuytren's contracture (Hurst, 1996).

The midaxial longitudinal incision above the palmar aspects of the digits, which should be always intersected by multiple Z-plasties at an angle of 60 degree to prevent scarring, may theoretically gain 75% of length (Lubahn, 1996). Larger Z angles, however, make flap transposition increasingly difficult. Smaller angles simplify flap transposition, but increase the risk of flap necrosis, because of the narrowed Z-plasty tips. That is why the flaps should be modified by favoring rounded incisions to broaden the flap tips.

The open palm technique requires primary closure of only longitudinal incisions. It is therefore important to place transverse incisions primarily

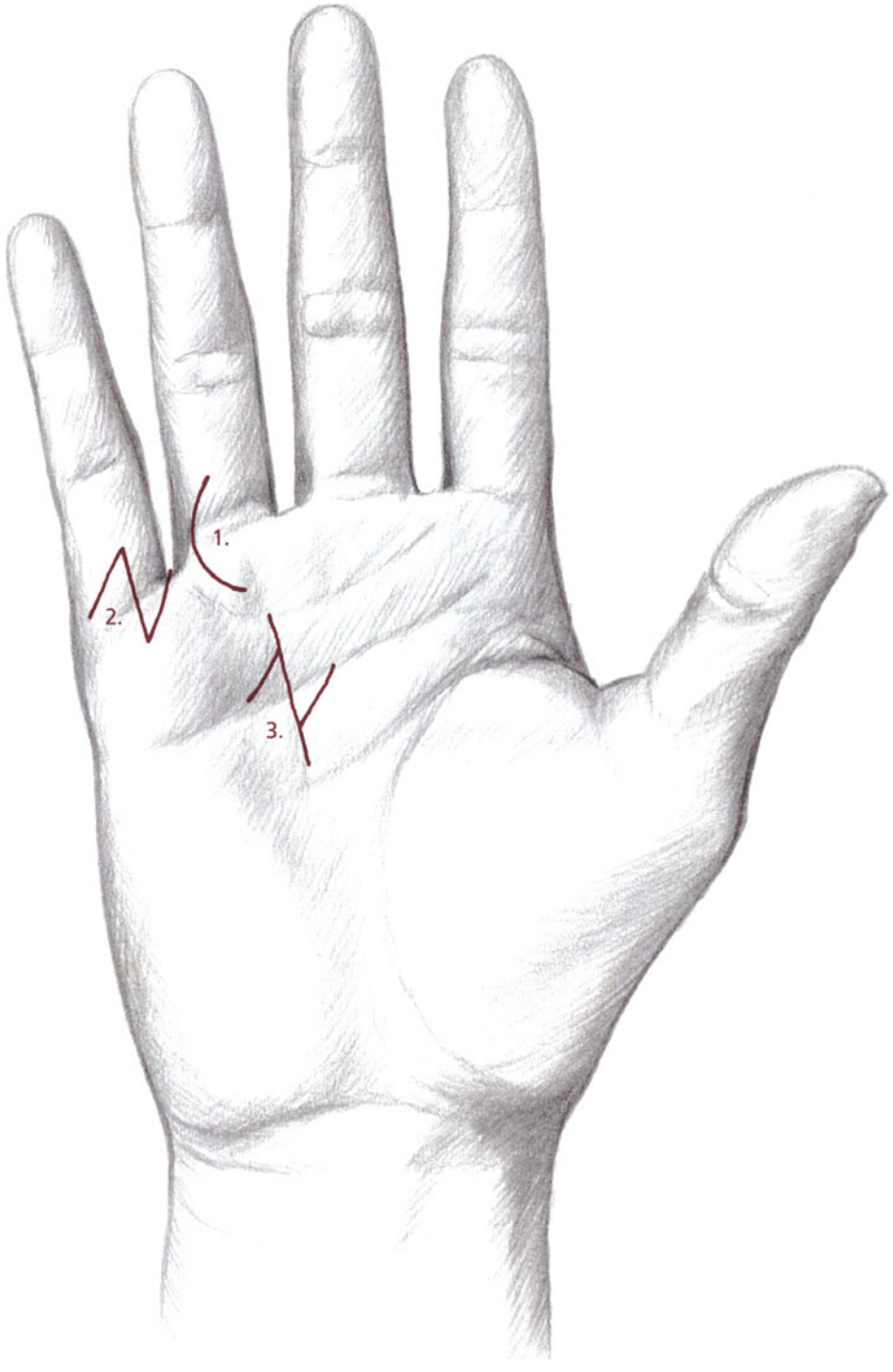


Fig. 39a. Minimal invasive access: (1) C-shaped incision for segmental fasciotomy according to Moermans. (2) Z-shaped limited incisions for fasciotomy. (3) small longitudinal incision interrupted by a Z-plasty, which is ideal for a limited fasciectomy.

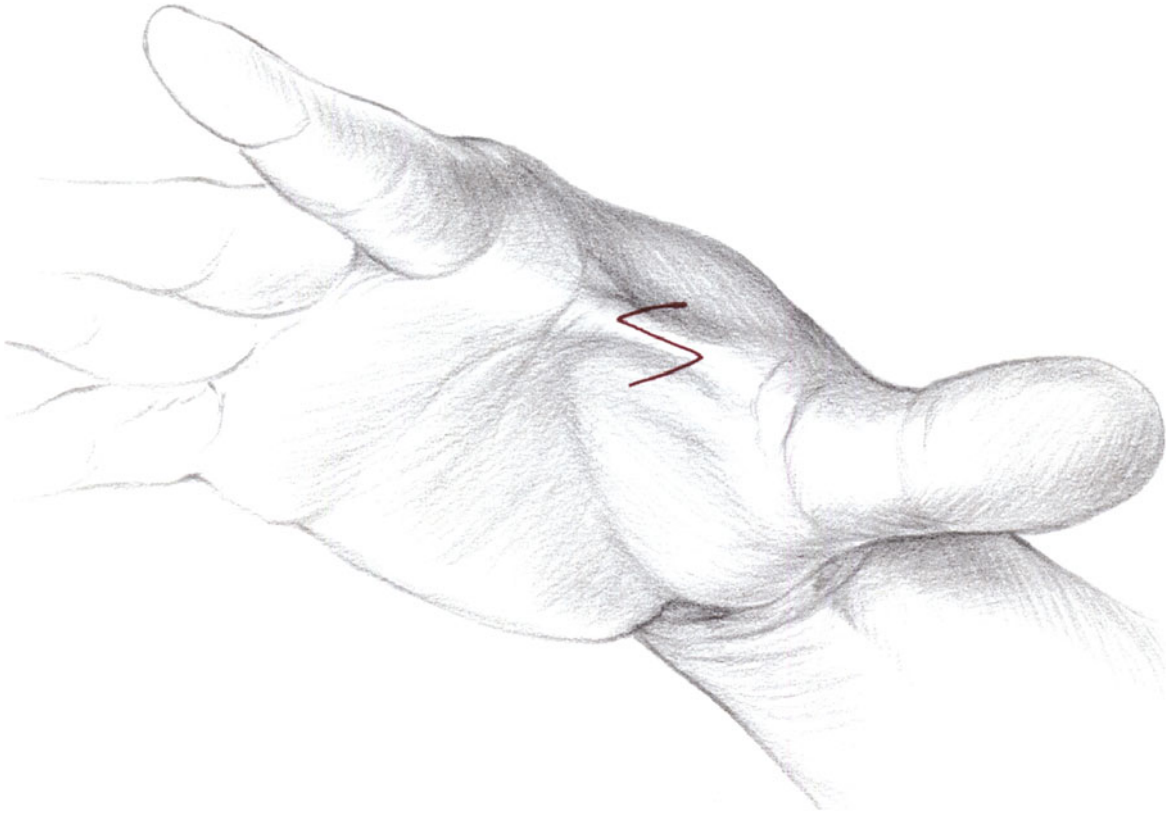


Fig. 39b. Outlined minimal invasive approach (primary Z-plasty flaps) in the first web space.

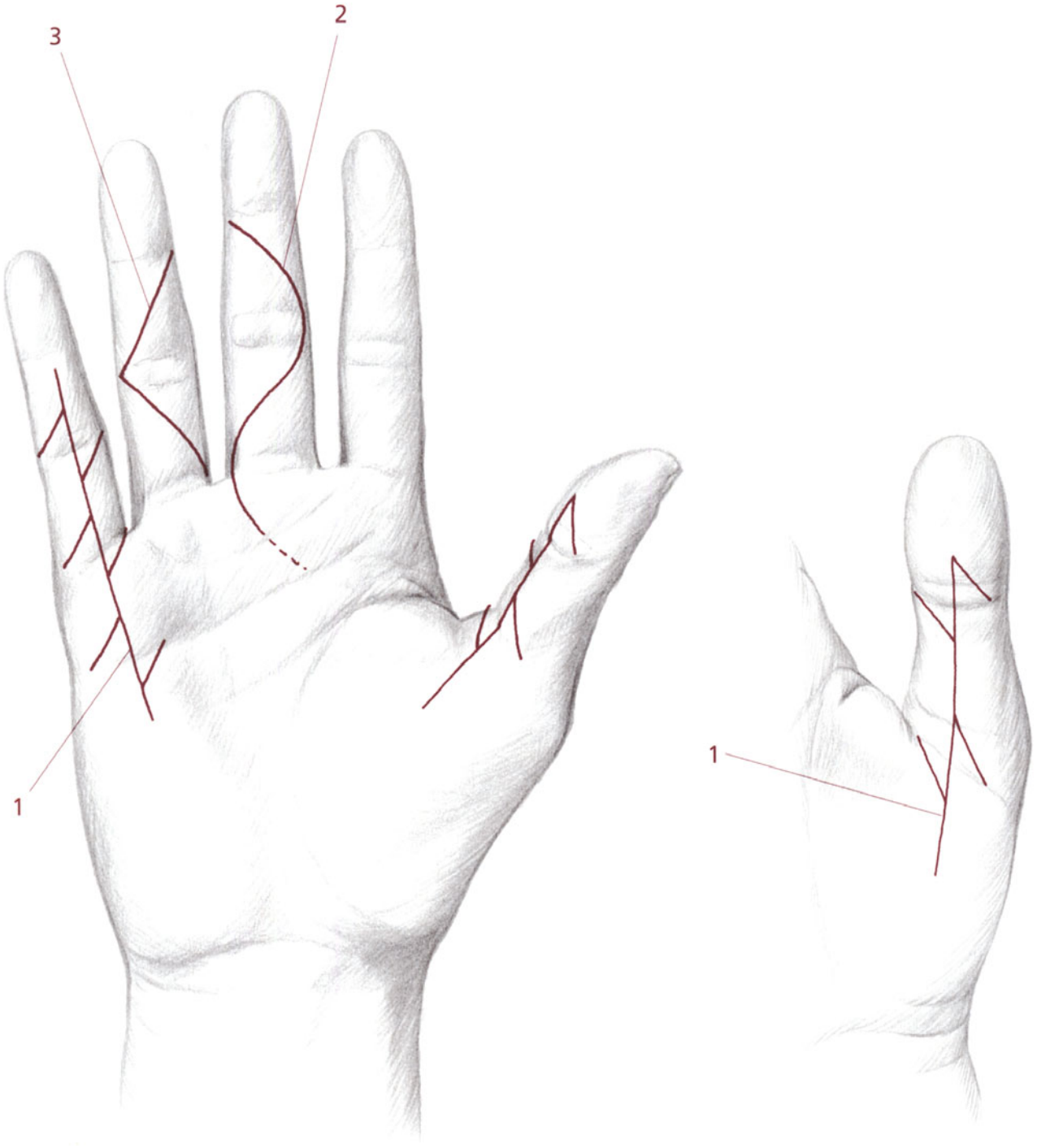


Fig. 39c. Basic features of the palmar longitudinal incisions: (1) straight incision with interrupted multiple Z-plasties on the thumb and little finger. (2) Serpentine-shaped longitudinal incision. Lazy-S scars tend to contract. (3) Zigzag Bruner incision.

in the palmar skin affected by the disease or in the palmar interphalangeal creases (Fig. 39d).

Many different palm incisions have been proposed (GELDMACHER, 1972; BRÜCKNER et al., 1986). We favor the Y-shaped incision because their vascular supply through their equally long hypothenar, thenar, or monticule bases is very reliable. This type of a triangular approach was appropriately described as a "Mercedes star-shaped" incision (MCGROUTHER, 1988). To assure the viability of these random pattern skin flaps, a sufficiently thick subcutaneous tissue layer needs to be maintained, which guarantees the vascular supply via the subdermal plexus (Fig. 39e).

According to TUBIANA et al. (1992), the main difference with respect to the required exposure for a planned fasciotomy versus a fasciectomy is in the extent to which the skin needs to be undermined. A fasciectomy will obviously necessitate more undermining.

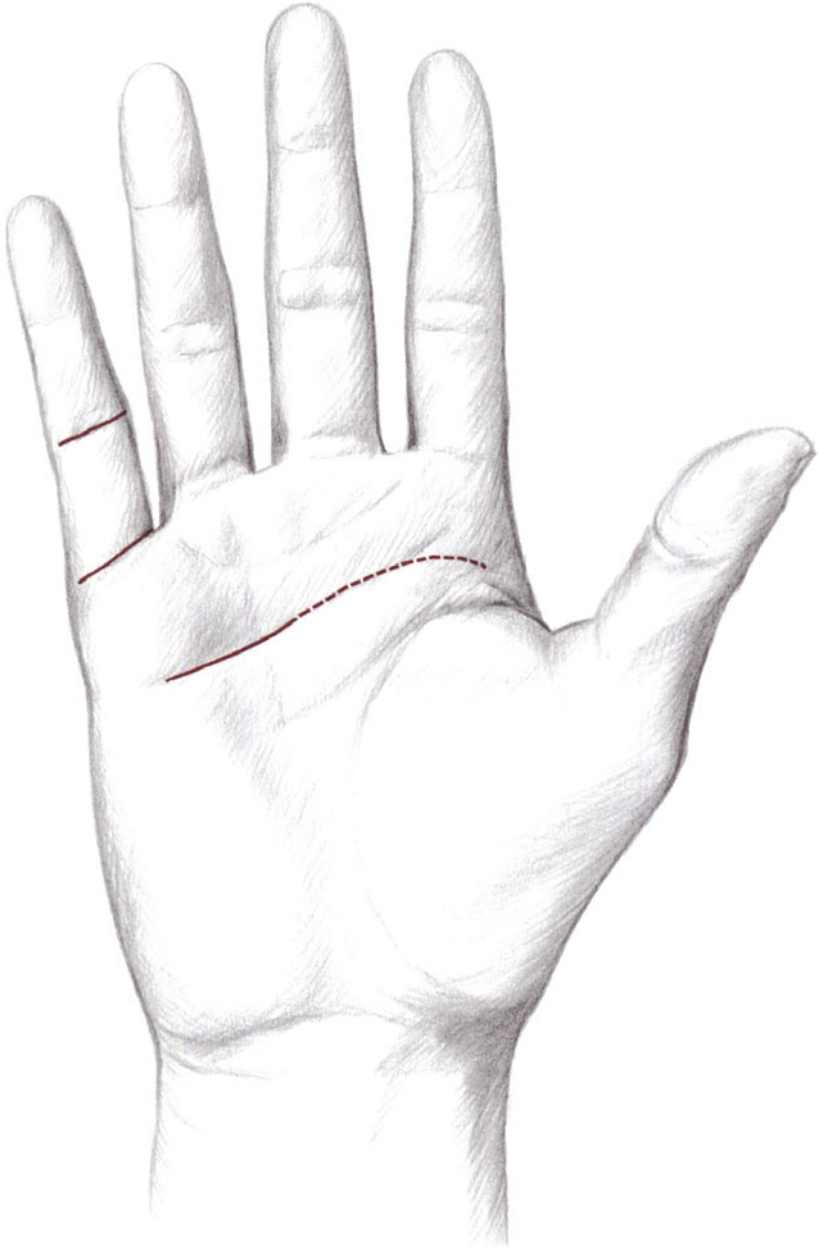


Fig. 39d. Transverse incisions between the proximal and distal palmar creases and in the digital flexion creases according to MCCASH.

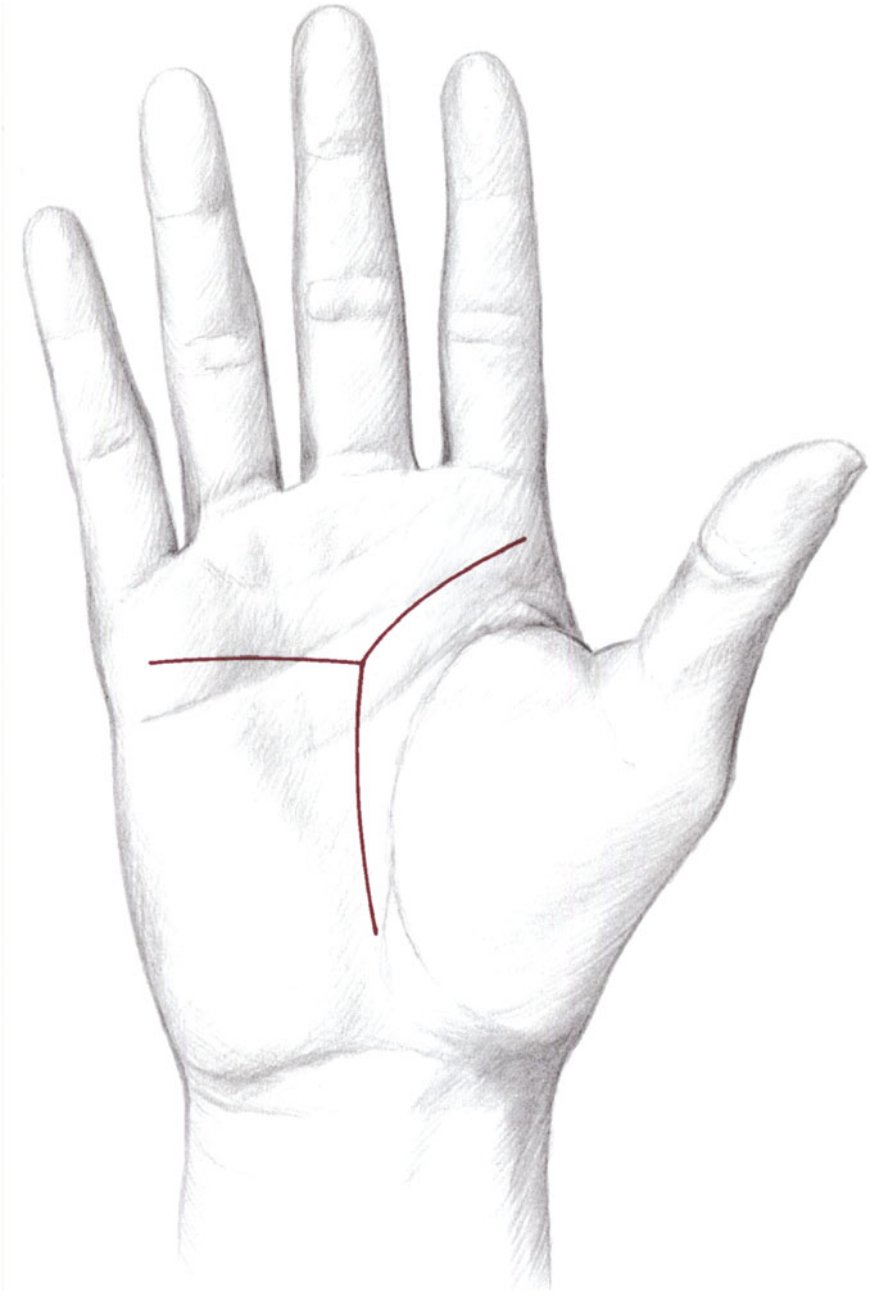


Fig. 39e. Mercedes star-shaped incision according to Millesi. The 120-degrees create three broad triangular flaps, which guarantee a secure blood supply via thenar, hypothenar and monticuli. Broad flap bases are the best prevention against apical necrosis.

20.3 Excision of the tissue affected by Dupuytren's disease

20.3.1 Fasciotomy

The oldest and most tissue-sparing procedure is the fasciotomy, which consists of the simple division of the diseased cords. Fasciotomies are mainly used in elderly patients and in those with multiple associated illnesses. In the past, a fasciotom, a curved sharp tool, was used. The surgeon performed this procedure blindly, and neurovascular lesions were frequent. The occasional fasciotomy done today is exclusively performed under vision as an open fasciotomy (BRENNER, 1997). Usually this procedure does not entail the removal of affected tissue (Fig. 40a–c).

A transverse incision of approximately 1–2 cm in the distal palmar crease above the hardened Dupuytren cord provides access, and sharp dissection continues towards the proximal and distal palm below the cutaneous/subcutaneous layer and on top of the palmar aponeurosis. Fine double skin hooks or traction sutures retract the edges of the incision to provide optimal exposure. The finger is extended and the cord severed with a #15 blade. A characteristic sound is heard when the cord is divided. After "redressement forcé" painstaking hemostatic measures are taken with bipolar cautery (Fig. 41a–41d).

Fig. 40a. Contracture of the ring and little finger corresponding Tubiana stage IV. The finger tips reaching into the hollow of the palm (beware: risk for pressure sores). The hyperextend DIP joints indicate that the dorsal digital fascial condensates are altered because of Dupuytren's disease. Minimal lateral access for a fasciotomy at the ulnar level of the neck of metacarpal V.

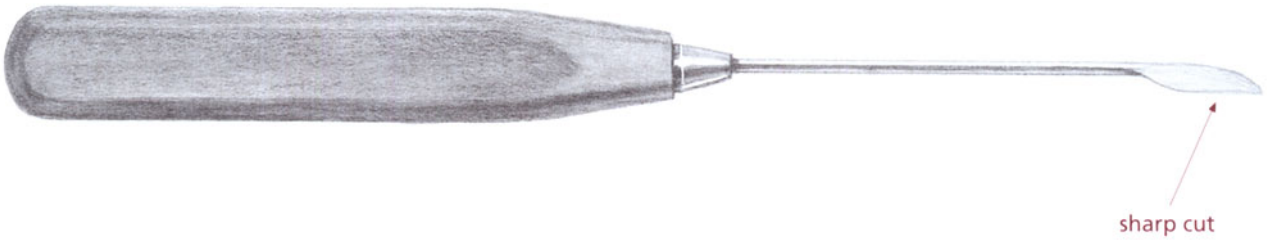


Fig. 40b. Extremely sharp fasciotom for blind subcutaneous cord division ("blind fasciotomy").

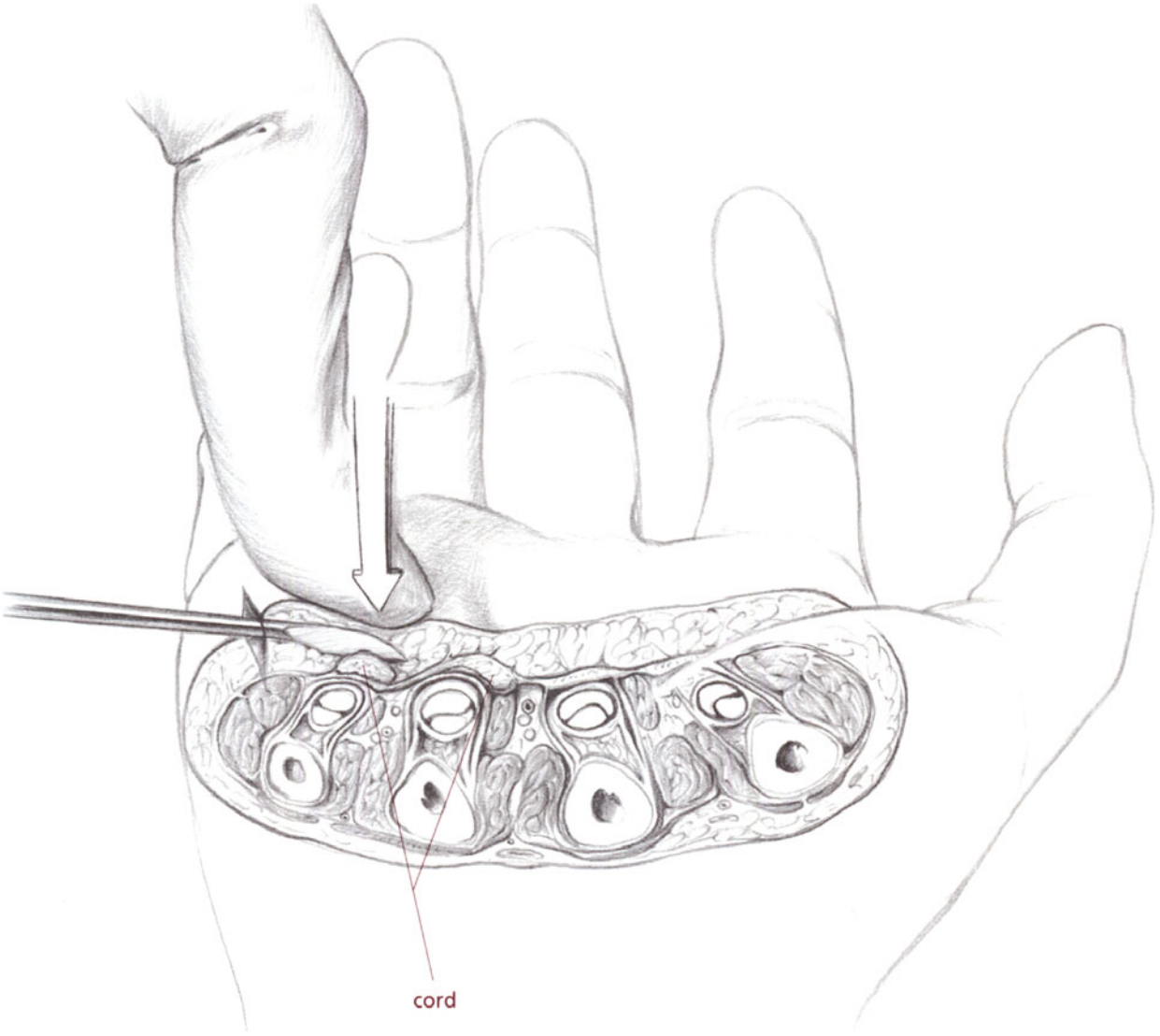


Fig. 40c. Traditional subcutaneous fasciotomy. From an incision on the ulnar surface of the distal hypothenar eminence, the fasciotom is introduced blindly and advanced under palpatory guidance towards the palm between the subcutaneous tissue and the diseased palmar aponeurosis. Firm pressure is exerted on the cords with the fasciotom, in order to divide them. Beware of risk of nerve damage from subcutaneous fasciotomy.

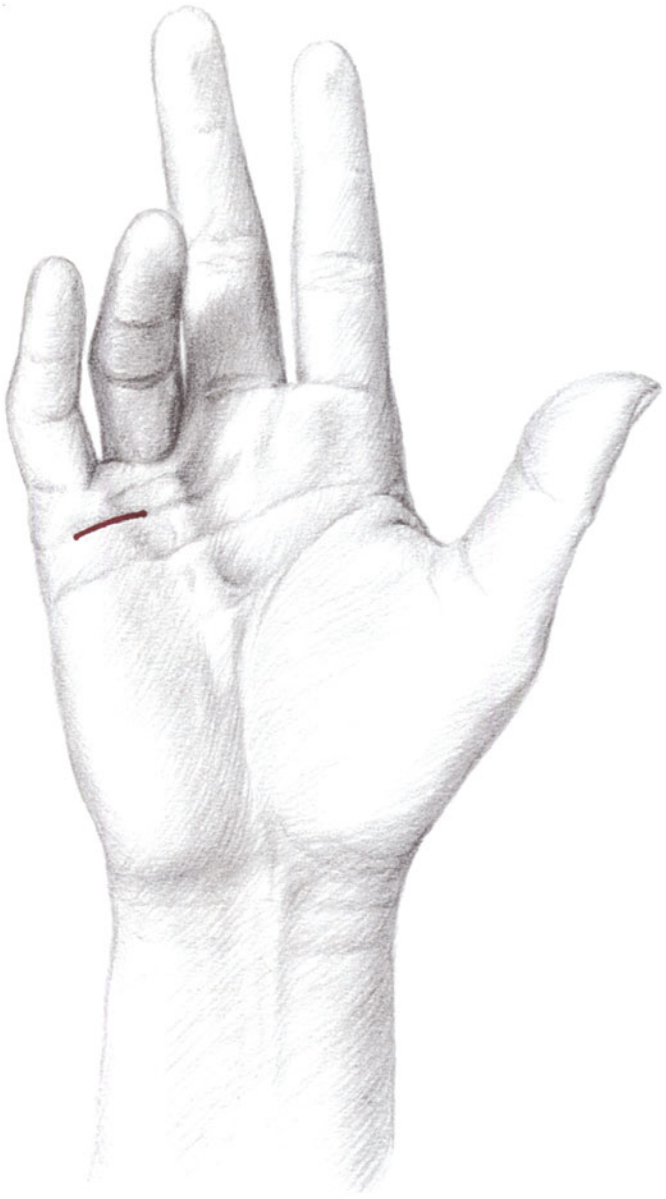


Fig. 41a. The principle of "open" fasciotomy under vision. A transverse 1–2 cm long incision in the distal palmar crease above the hardened cord is a suitable approach if two or more finger rays are affected by Dupuytren's disease.

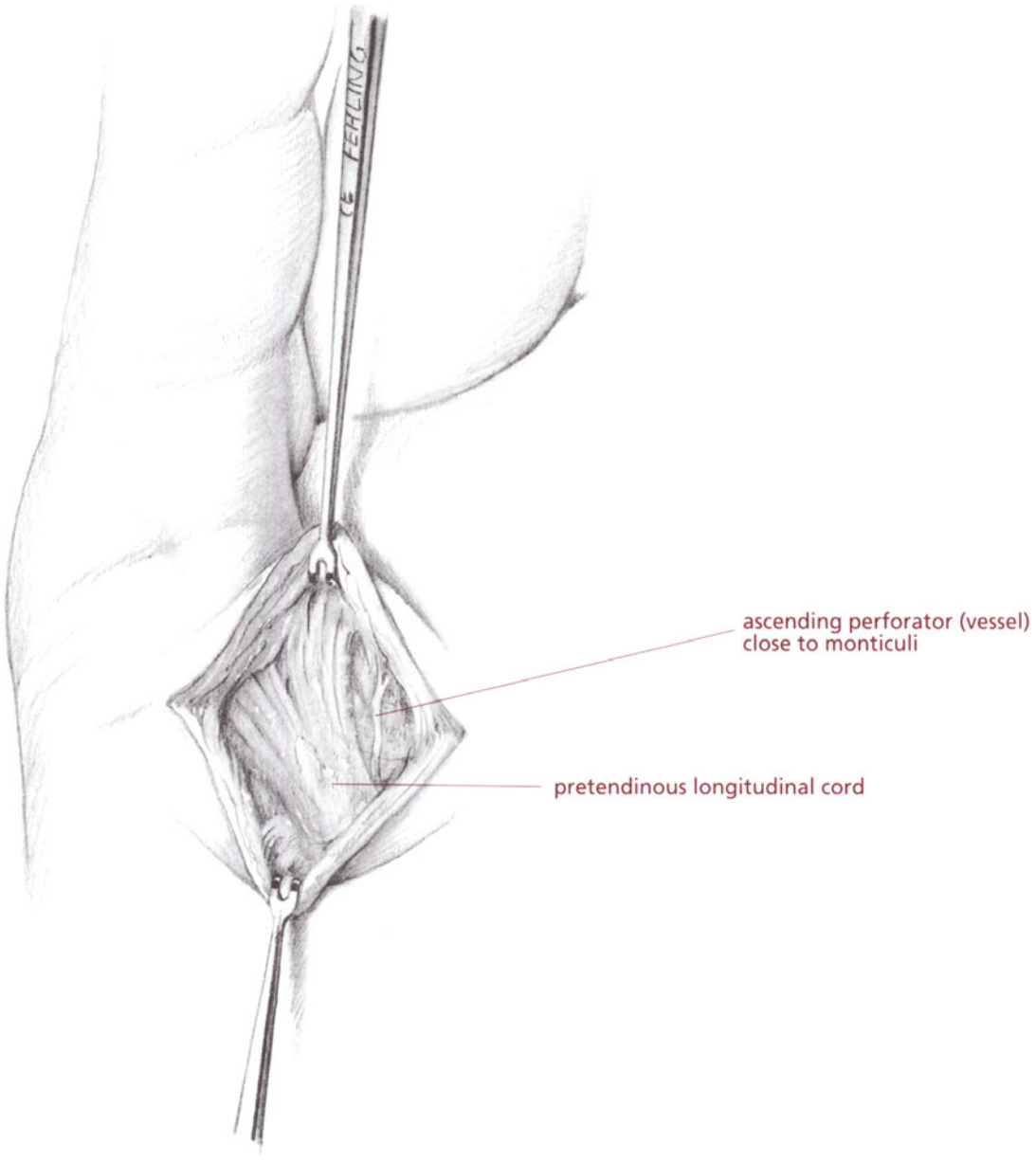


Fig. 41b. Sharp dissection of the proximal and distal parts of the palmar aponeurosis between subcutaneous tissue and aponeurosis. Fine double skin hooks or traction sutures hold the wound margins apart to permit visualization on the operating field.

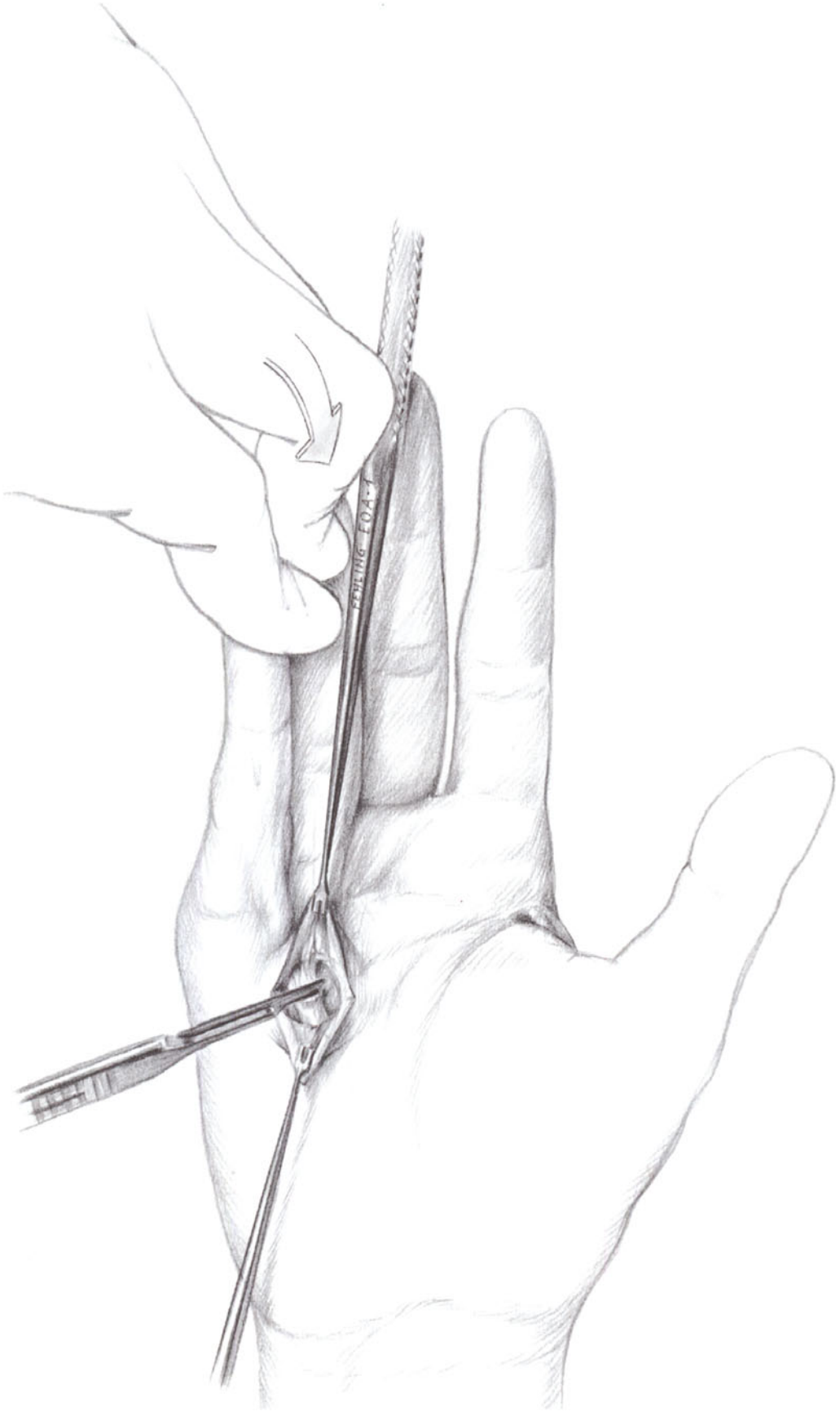


Fig. 41c. The contracted finger is extended and a characteristic sound is audible when the cord is divided under moderate scalpel pressure. After redressement forcé, subtle hemostasis is achieved by bipolar cautery.

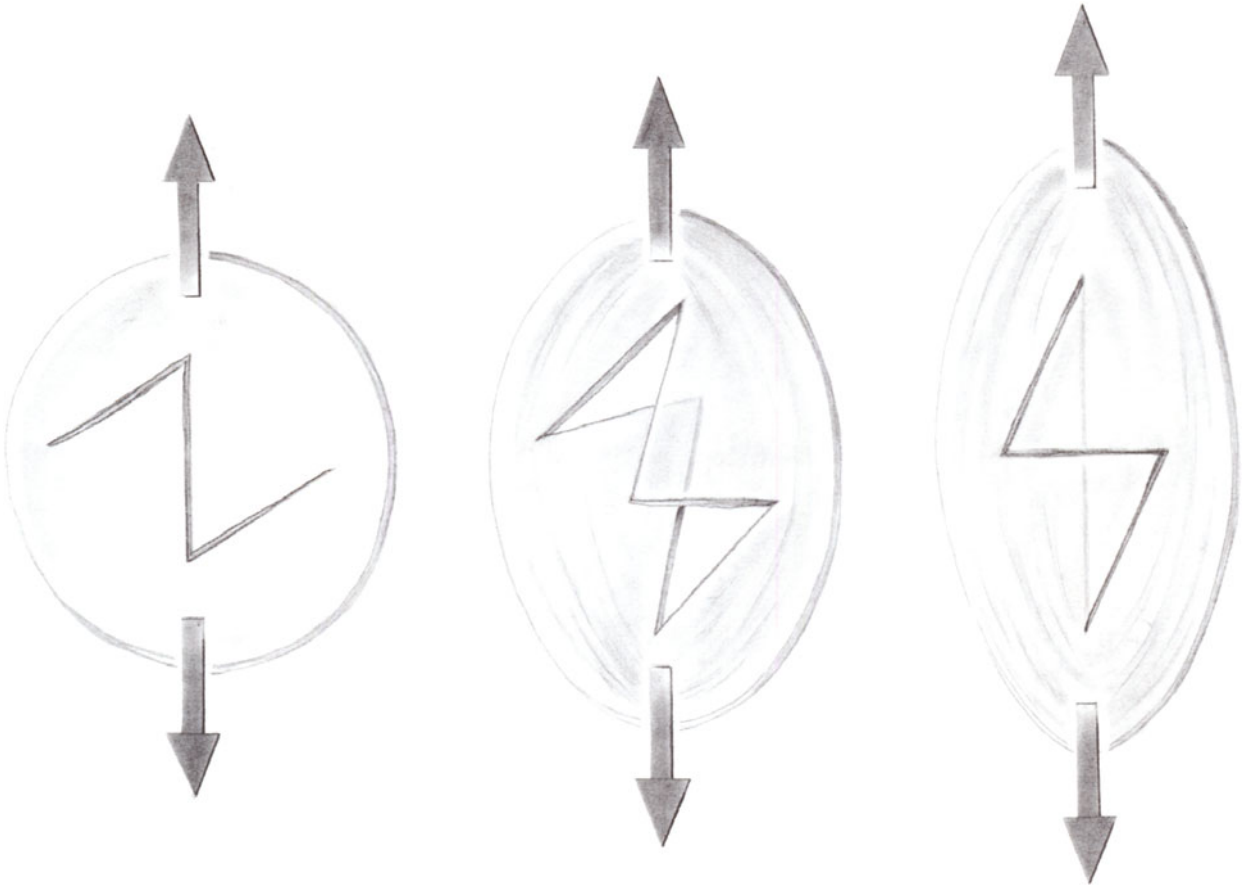


Fig. 41d. Instead of a small transverse distal palmar crease incision, a primary and straight longitudinal access followed by immediate Z-plasties can be used for an "open" fasciotomy. The arrows symbolize the traction due to finger extension. From the left to right, the illustrations depict that any actual length gained has been achieved at the expense of the breadth of the digitopalmar soft tissues.

20.3.2 Needle "fasciectomy"

Closed needle fasciectomy, an outpatient procedure, is also performed under loco-regional anesthesia in a bloodless field. The 16-gauge cannula used to inject the anesthetic is applied to the edge of the palpable cord in a "sawing" motion from the dorsal to the palmar side until the cord snaps when the affected finger is suddenly extended ("redressement forcé") (Fig. 42). This procedure is preferably performed in the palm, above the metacarpophalangeal joints, the proximal and—more rarely—the middle phalanx. Needle "fasciectomy," which in reality is a special form of fasciotomy, is never used on the distal phalanx (LERMUSIAUX et al., 1997).

After forced extension of the finger a sparing dressing is applied. A suture is unnecessary.

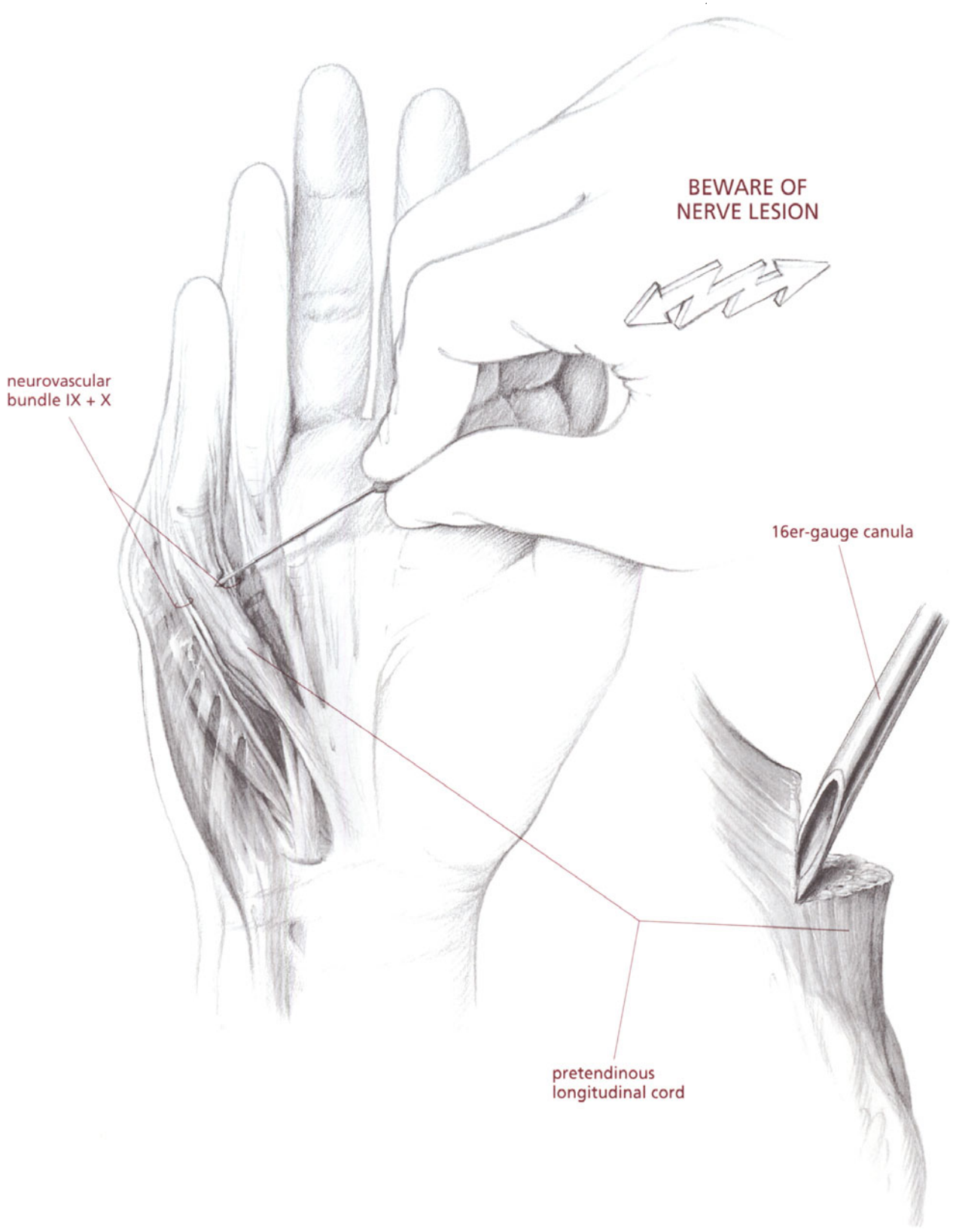


Fig. 42. For the so-called needle fasciectomy, which is actually a form of fasciotomy and represents a palliative procedure, the needle used to administer local/regional anesthesia is also used as a cutting device for "cordotomy". The needle tip is moved in a sawing motion against the fibrous cord in dorso-palmar direction. This motion is repeated several times. The contracture is finally released by forceful extension of the diseased finger. On the one hand, the "arrow" symbolizes the direction of motion, on the other, it indicates areas of potential nerve damage.

20.3.3 Segmental aponeurectomy

Minimal invasive segmental aponeurectomy is the limited removal of a defined cord, which blocks extension. This is done through a small incision, which can be C-shaped and about 1.5 cm long. Alternatively, a Z-shaped incision can be made immediately, or one can use a longitudinal (on top of the highest point of the cord) incision with broken secondary Z-plasties. The limbs of the flaps are always situated outside the affected region. One should follow MOERMANS' (1991) recommendation and dissect very close to the dermis, to avoid any bridges to the fascia. The incision extends to the diseased palmar fascia. During the whole maneuver an assistant keeps the contracted finger under tension. Small Gillies- and double skin hooks retract the skin edges. This provides a clear view of the border between the tissue affected by Dupuytren's disease and the macroscopically unaffected areas of the palmar fascia. No dissection or resection is performed beyond this margin. Wide undermining is therefore avoided. Only affected tissue of the palm or finger is resected, after the bilateral neurovascular bundles are identified (Fig. 43a–e). One needs to be aware of

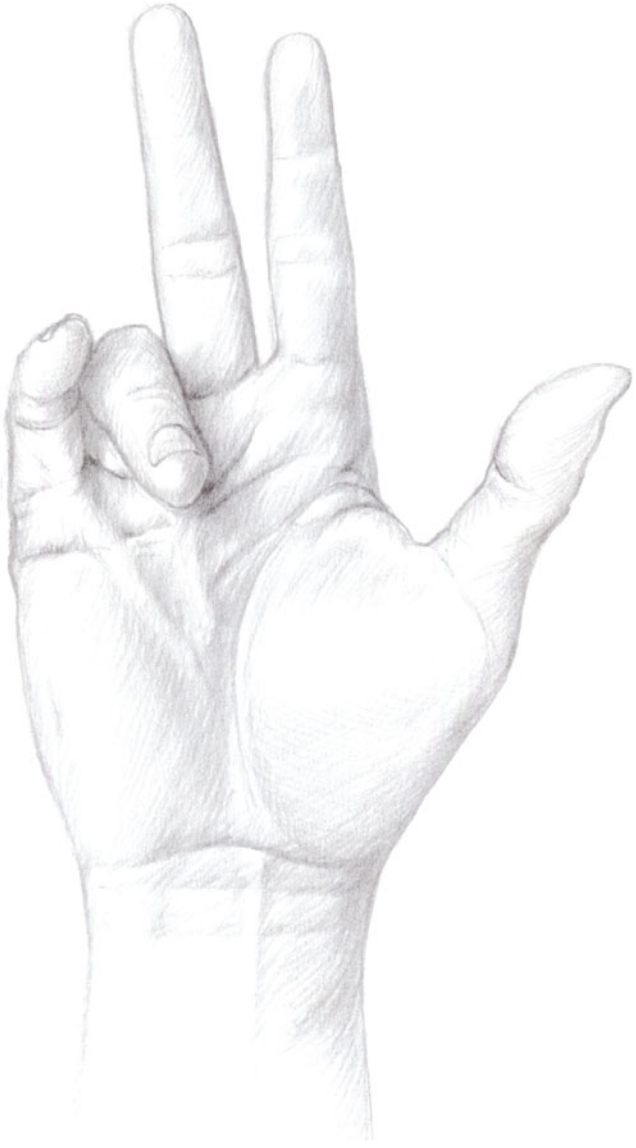


Fig. 43a. Tubiana stage III flexion contracture affecting ring finger and little finger (initial clinical finding).

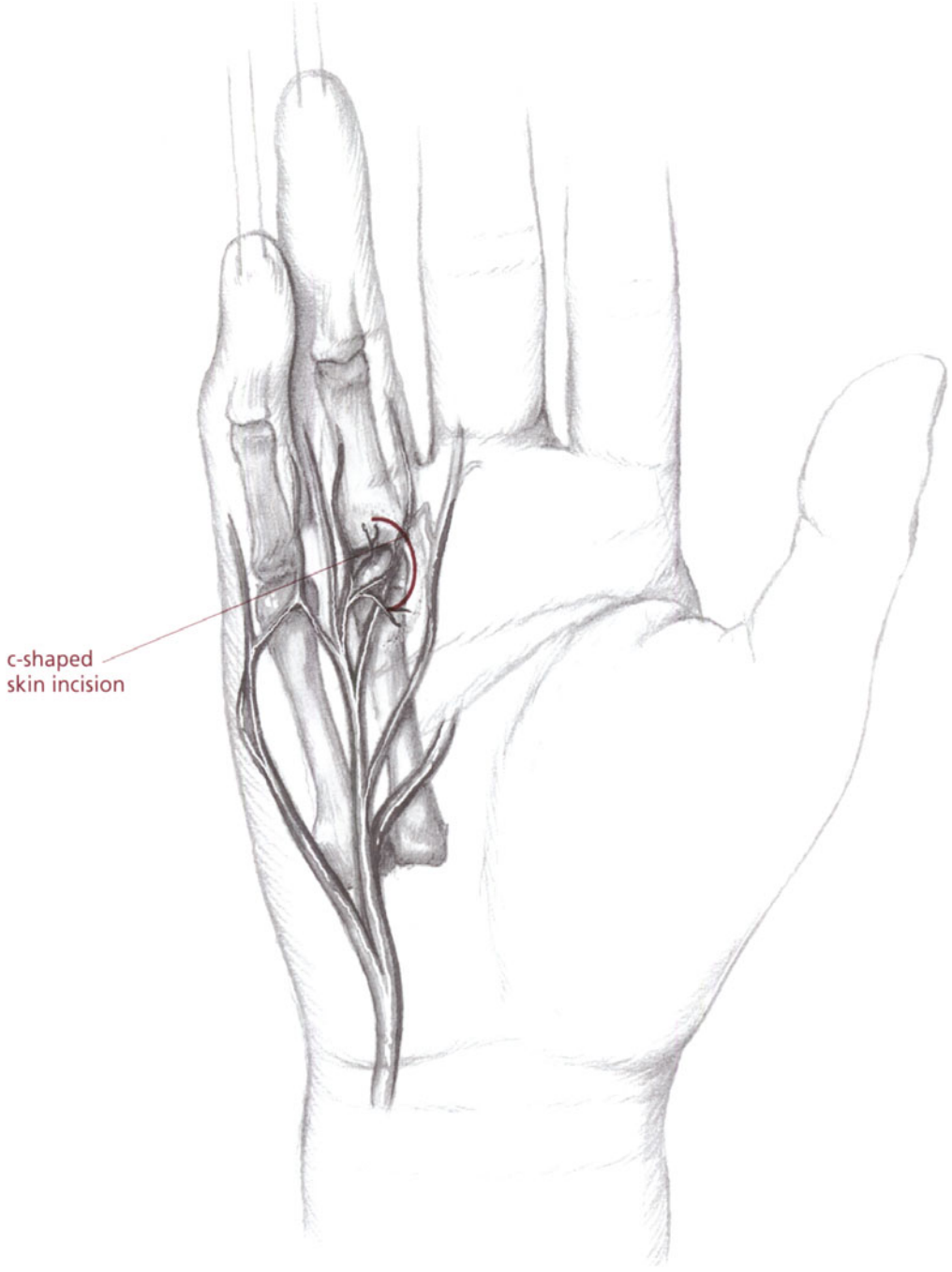


Fig. 43b. Small C-shaped skin incision around the area that has fibromatous involvement.

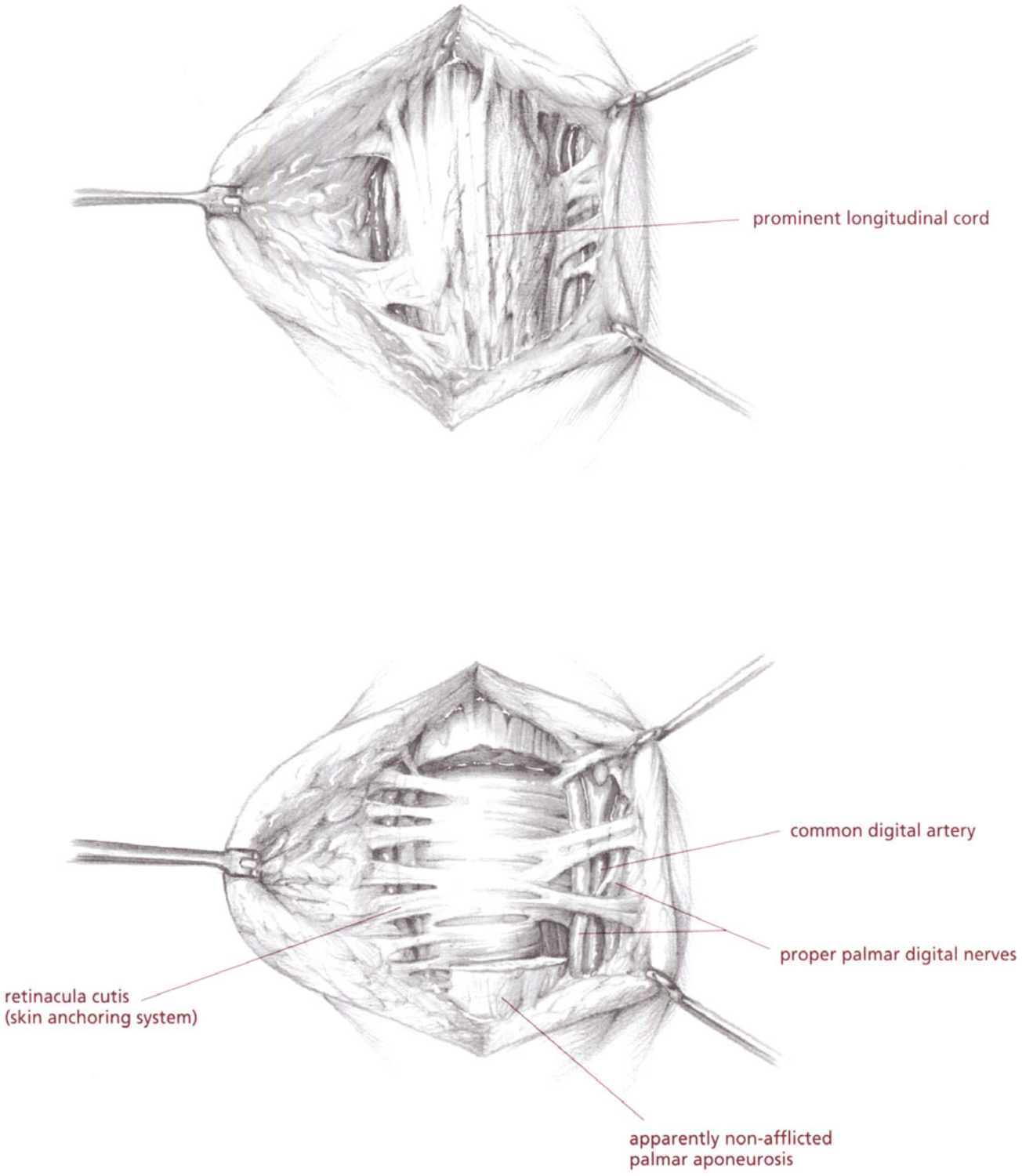


Fig. 43c, d. Dissection of the skin/subcutaneous layer and exposure of the diseased palmar fascia. Stamp-sized excision of the diseased tissue without safety margin. The site is clearly visible and the neurovascular bundles are therefore safe.

Fig. 43e. Wound closure with interrupted stitches after segmental fasciectomy

displaced neurovascular bundles in cases of digital spiral cords. Exposure of a displaced corkscrew-like neurovascular bundles is always more risky than that of a straight, non-displaced bundle. The removed diseased fascial tissue is kept for histological analysis. Usually, functional improvement can be observed immediately after segmental aponeurectomy.

A Penrose drain and compression dressing are advised.

According to MOERMANS (1991), residual PIP contractures are best treated by postoperative mobilization and not by capsulotomy.

20.3.4 Partial fasciectomy

Traditional partial fasciectomy includes resection of the fibrous diseased cord together with the macroscopically unaffected fascial tissue, including a small margin of approximately 0.5 cm (BRENNER, 1997).

The resection is much quicker in partial than in complete fasciectomy. Both the recurrence rates and the extension, which can be accomplished, seem almost identical, however.

A midaxial longitudinal incision is made on the contracted finger, sparing the distal interphalangeal joint. Although Z-flaps are not made until after the fasciectomy, they are designed and marked at this early stage. It is important to consider the possibility of unintentional skin perforations ("button holes") and disturbed vascular supply of the flaps. The degree of extension is determined next, before the flexed fingers are held with traction sutures placed through the fingertip or through the free border of the nail.

BRUNER incisions are only justified if the contracture is at least moderate (Fig. 44a–m).

To obtain maximal finger extension, a hand plate with holes is used. Elastic reins are passed and secured. The distal part of the finger can alternately be stretched by means of traction sutures. Exposure is via a midaxial longitudinal incision, which is deepened until fibromatous tissue is reached. Scalpel dissection is mandatory for this. Bilateral cutaneous/subcutaneous flaps are raised by careful dissection beyond the diseased palmar aponeurosis or digital cords. Stay sutures or hooks are placed. Subcutaneous veins or ascending perforators are coagulated with bipolar tweezers.

The healthy palmar aponeurosis is opened by exerting gentle pressure with a #15 blade, keeping a 5 mm safety margin. If the superficial palmar arch or the common neurovascular bundles are not visible right away, because their fibrofatty tissue envelope hides them, then the lobular layer is split lengthwise with scissors, starting from healthy tissue. The proximal, usually triangular part is grasped with Adson forceps and placed under tension, facilitating the division of the tensed tissue with the scalpel.

In the palm the procedure is performed from the radial to the ulnar side. The common neurovascular bundle is radially retracted together with the palmar lumbrical muscles by a blunt two-pronged retractor. The vertical septum between middle and ring finger becomes visible. This is resected at its deepest point with the scalpel by applying just the right pressure. The dissection

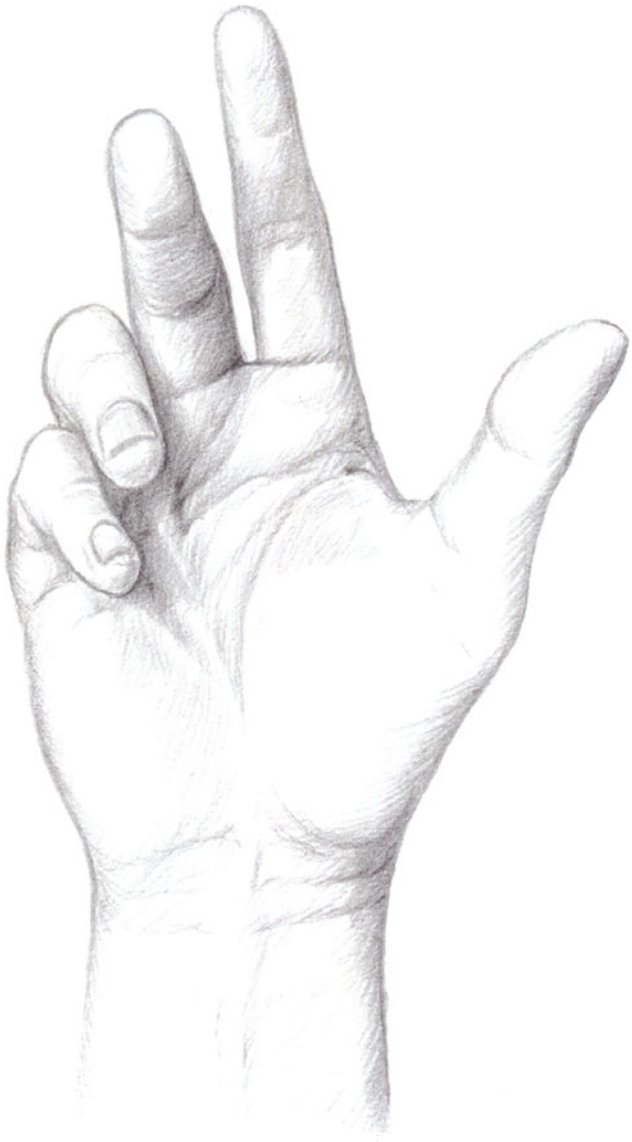


Fig. 44a. Partial fasciectomy. Dupuytren's contracture of ring finger and little finger resembling those of a violin player (stage IV).

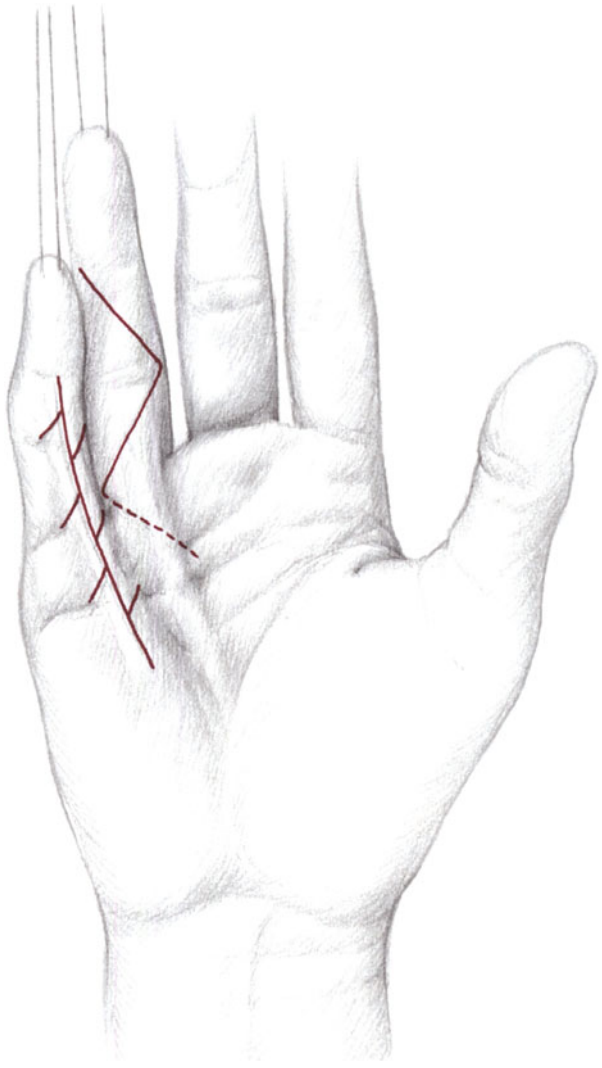


Fig. 44b. Longitudinal midline skin incision on the flexor side of the little finger, sparing the distal interphalangeal joint. In spite of the fact that the Z-plasties will not be cut until after the fasciectomy, they are already marked in this figure. When proceeding with Z-plasties, one has to consider accidental skin perforations ("button holes") and possibly impaired blood supply to the flaps. After the extension capacity has been determined, the flexed fingers are fixated by traction sutures either through the tip or the free border of the nail. On the ring finger zigzag right-angled Bruner incisions are visible. These are justified only in moderate forms of contracture.

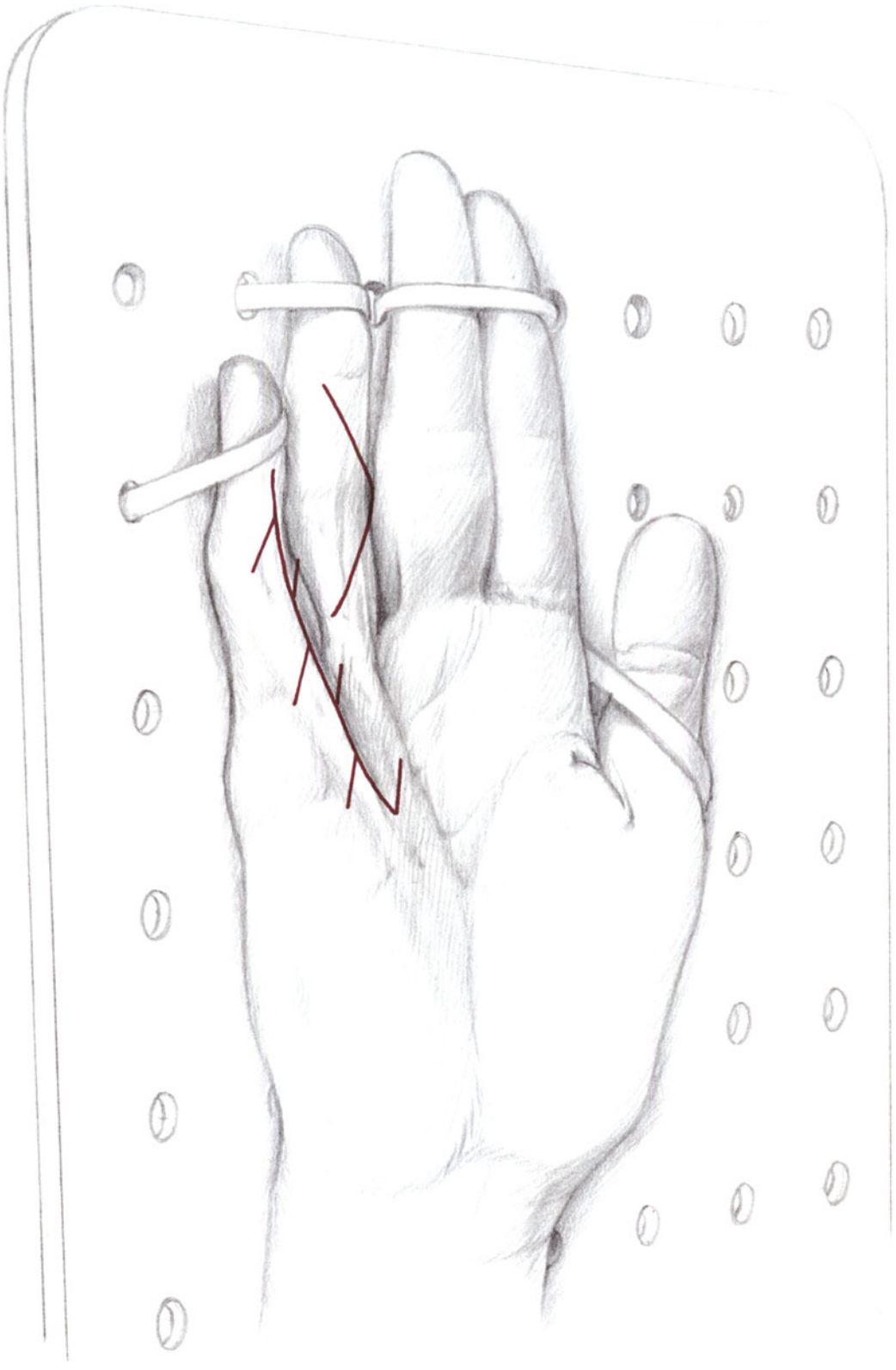


Fig. 44c. To obtain maximal finger extension, a hand plate with holes is used. Rubber bands are passed through these holes and tightened on the reverse side with Kocher clamps. The incision can be extended towards the palm either in an axial direction or in the shape of a bayonet. Part of the latter has to follow the skin creases.

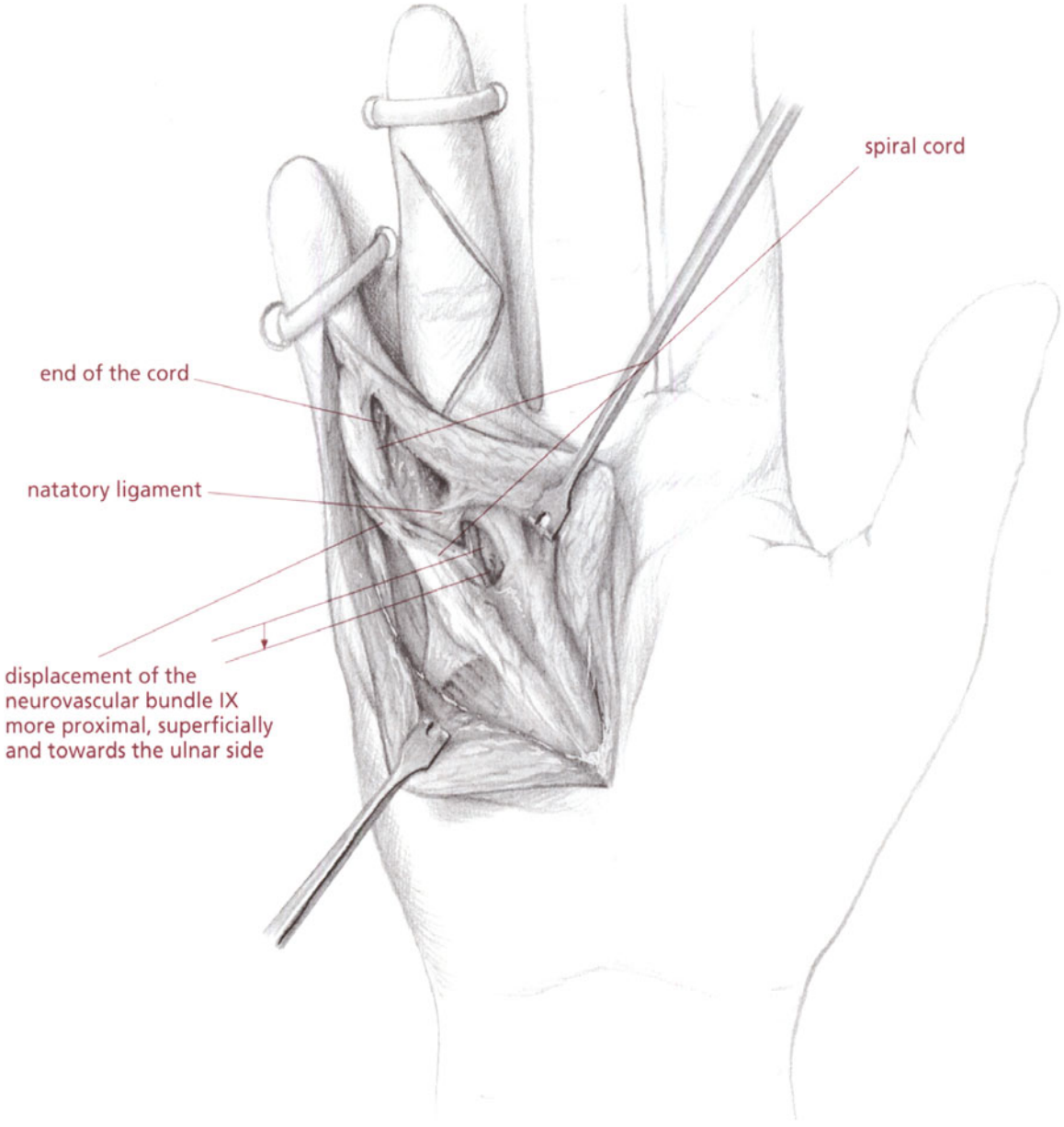


Fig. 44d. Digital longitudinal midline incision through skin and subcutaneous tissue and deepened to the fibromatous tissue. Remarkable is the dislocation of the neurovascular bundle to the proximal-ulnar site and superficially. Dissection is always performed with the scalpel.

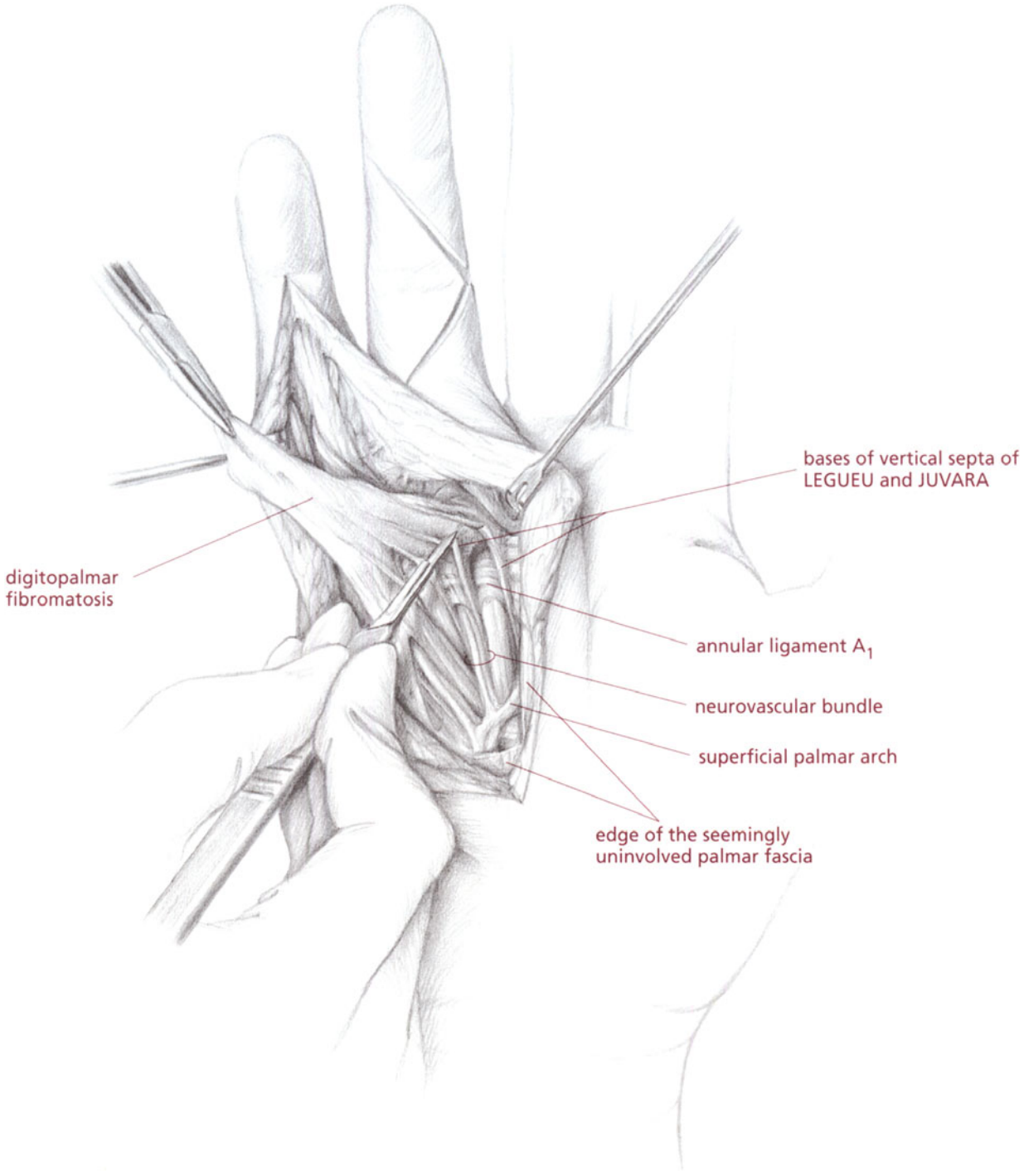


Fig. 44e. Meticulous bilateral undermining of the skin/subcutaneous flaps beyond the limits of the diseased palmar aponeurosis or the digital cords. Retraction with stay sutures or small hooks. Visible subcutaneous veins or ascending perforators are cauterized.

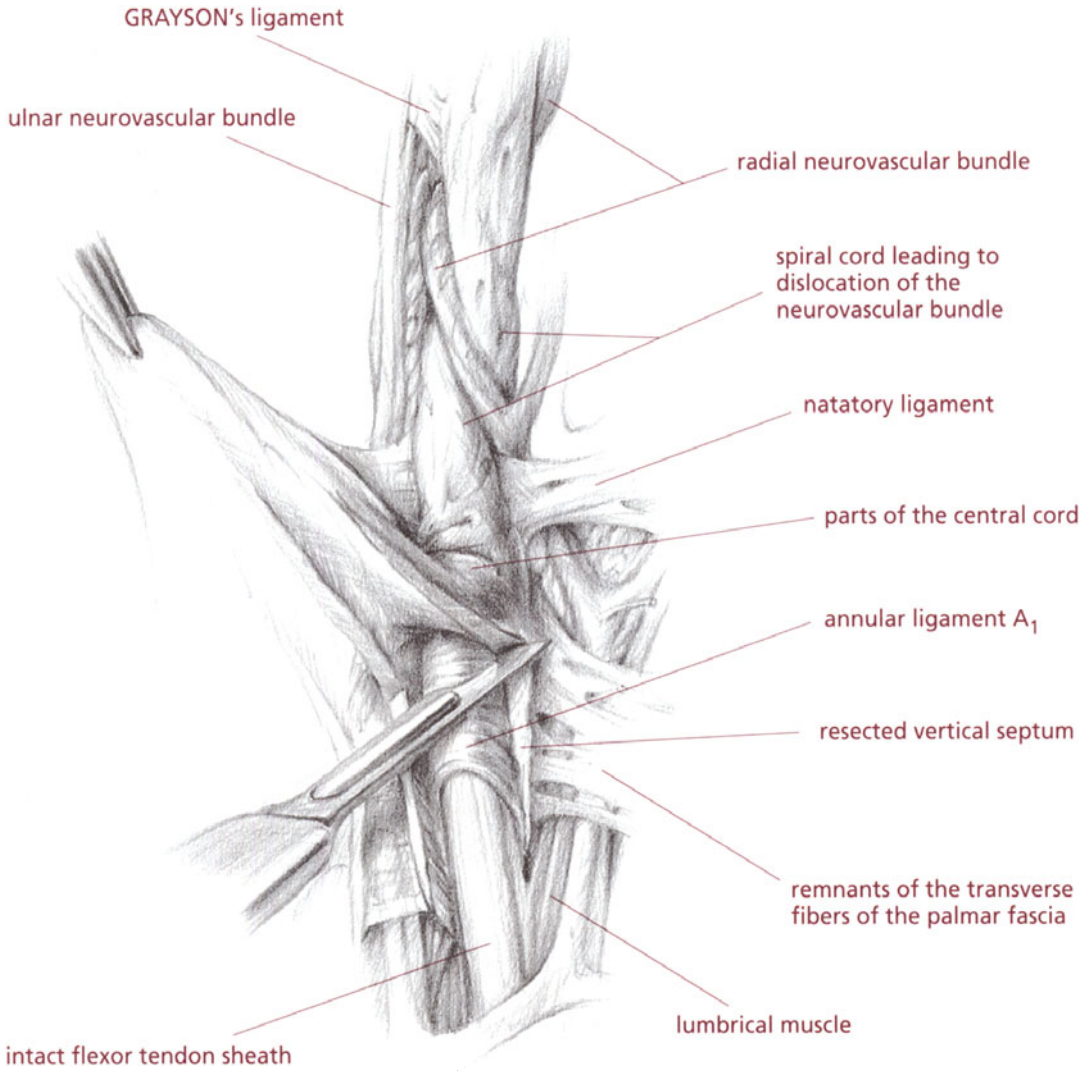

GRAYSON's ligament
ulnar neurovascular bundle
radial neurovascular bundle
spiral cord leading to dislocation of the neurovascular bundle
natatory ligament
parts of the central cord
annular ligament A_1
resected vertical septum
remnants of the transverse fibers of the palmar fascia
lumbrical muscle
intact flexor tendon sheath

Abb. 44 f. Fibromatous central and spiral cord, which cause pronounced flexion contracture of the proximal interphalangeal joints. Antegrade dissection of the palmar aponeurosis under tension, superficial to the intact neurovascular bundles and flexor tendon sheath including the pulleys. The vertical septa are included in the excision of the diseased fascial tissue. The dissection advanced from radial to ulnar. The safety margin to apparently uninvolved fascial tissue of the remaining palmar aponeurosis is 5 mm.

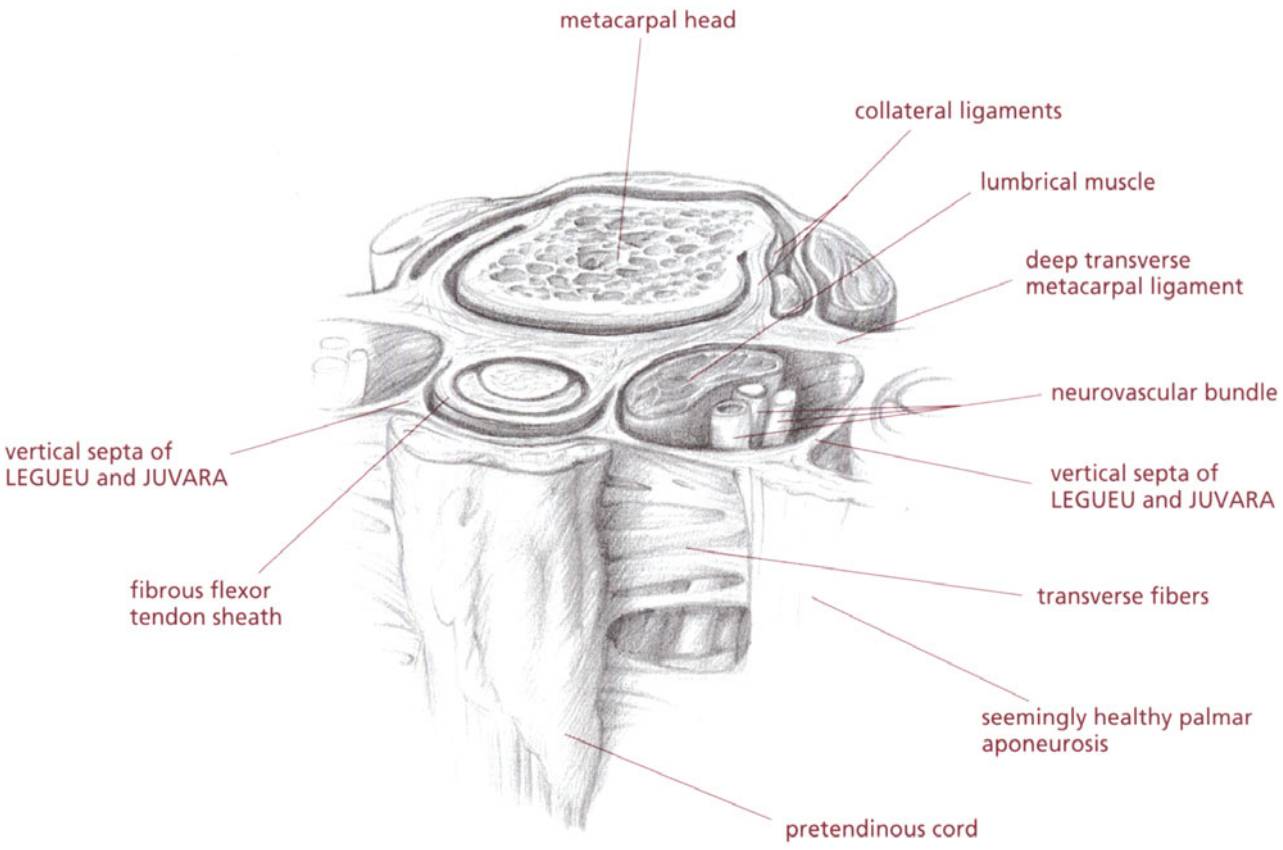


Fig. 44g. Transverse diagram of fascial elements at the subcapital level.

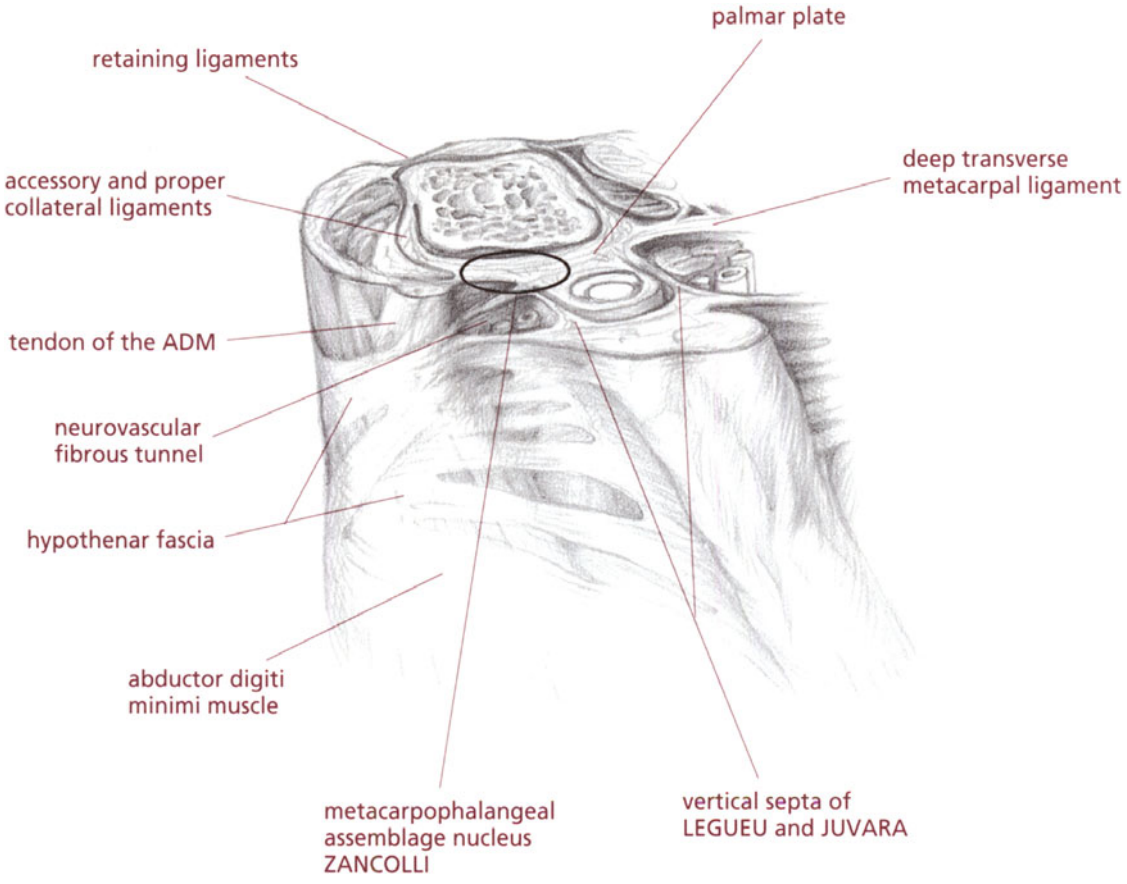


Fig. 44h. Detailed cross-sectional anatomy of fascial elements and tendons on the ulnar side.

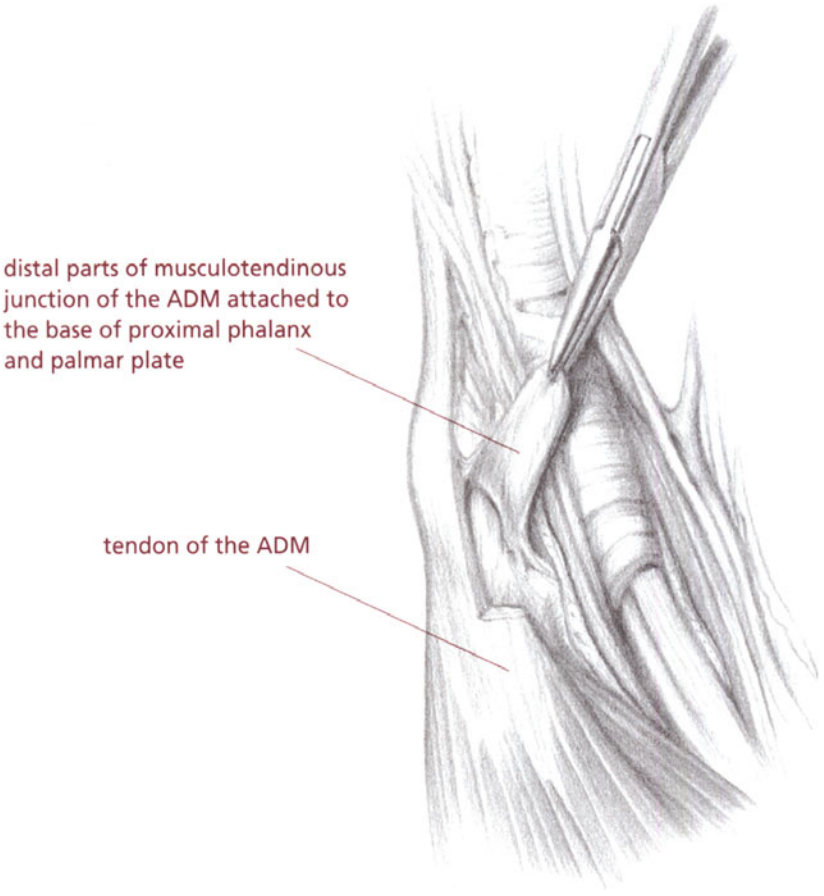

distal parts of musculotendinous junction of the ADM attached to the base of proximal phalanx and palmar plate
tendon of the ADM

Fig. 44i. Additionally, we perform a tenotomy of the abductor digiti minimi tendon close to the metacarpophalangeal assemblage nucleus of Zancolli ("Carrefour fibrieux"). This permits favorable extension of the proximal interphalangeal joint of the little finger. The latter maneuver is always a criterion for a successful fasciotomy of the ulnar side of the hand.

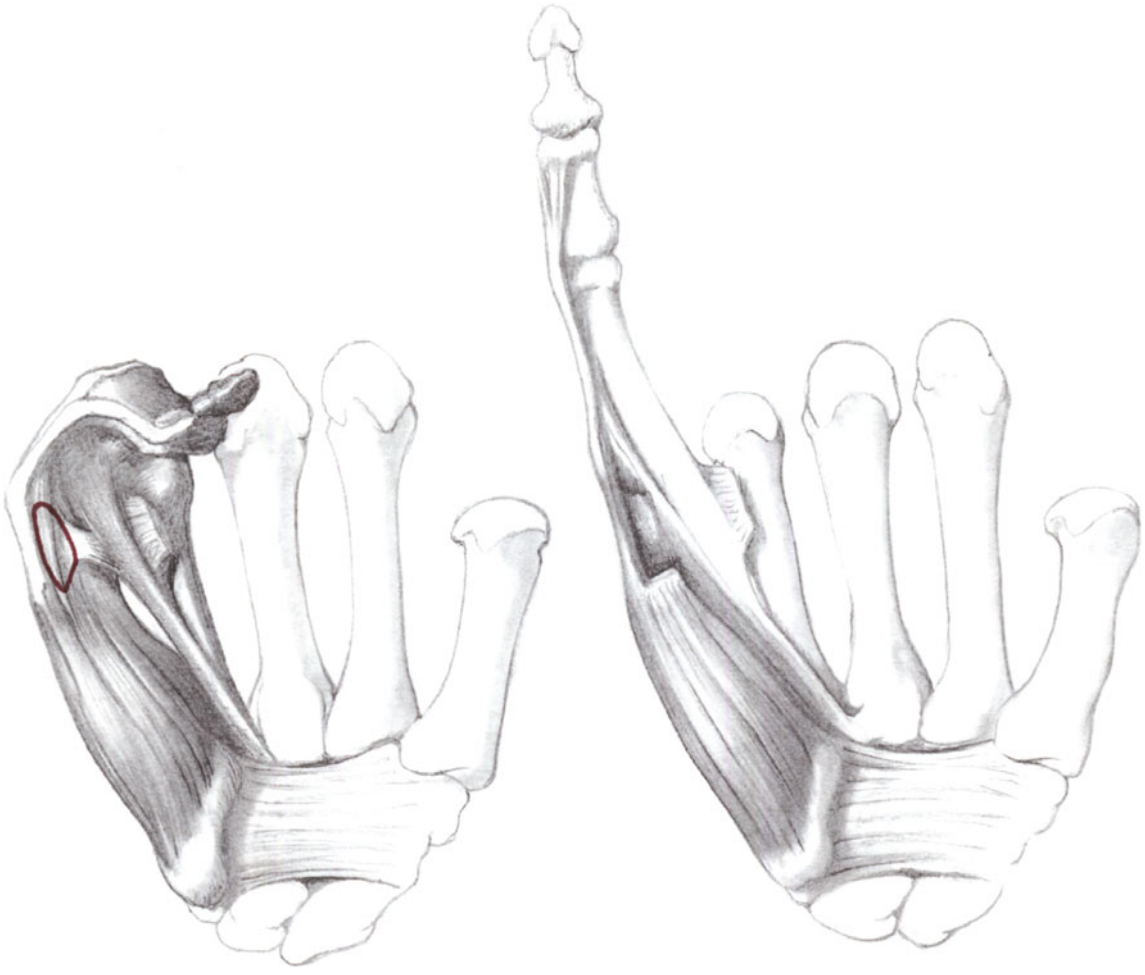


Fig. 44j. Effect of the abductor digiti minimi tenotomy on the palmar plate, the proximal phalanx as well as on the PIP joint. Left: state of contracture prior to tenotomy. Right: nearly full extension following tenotomy.

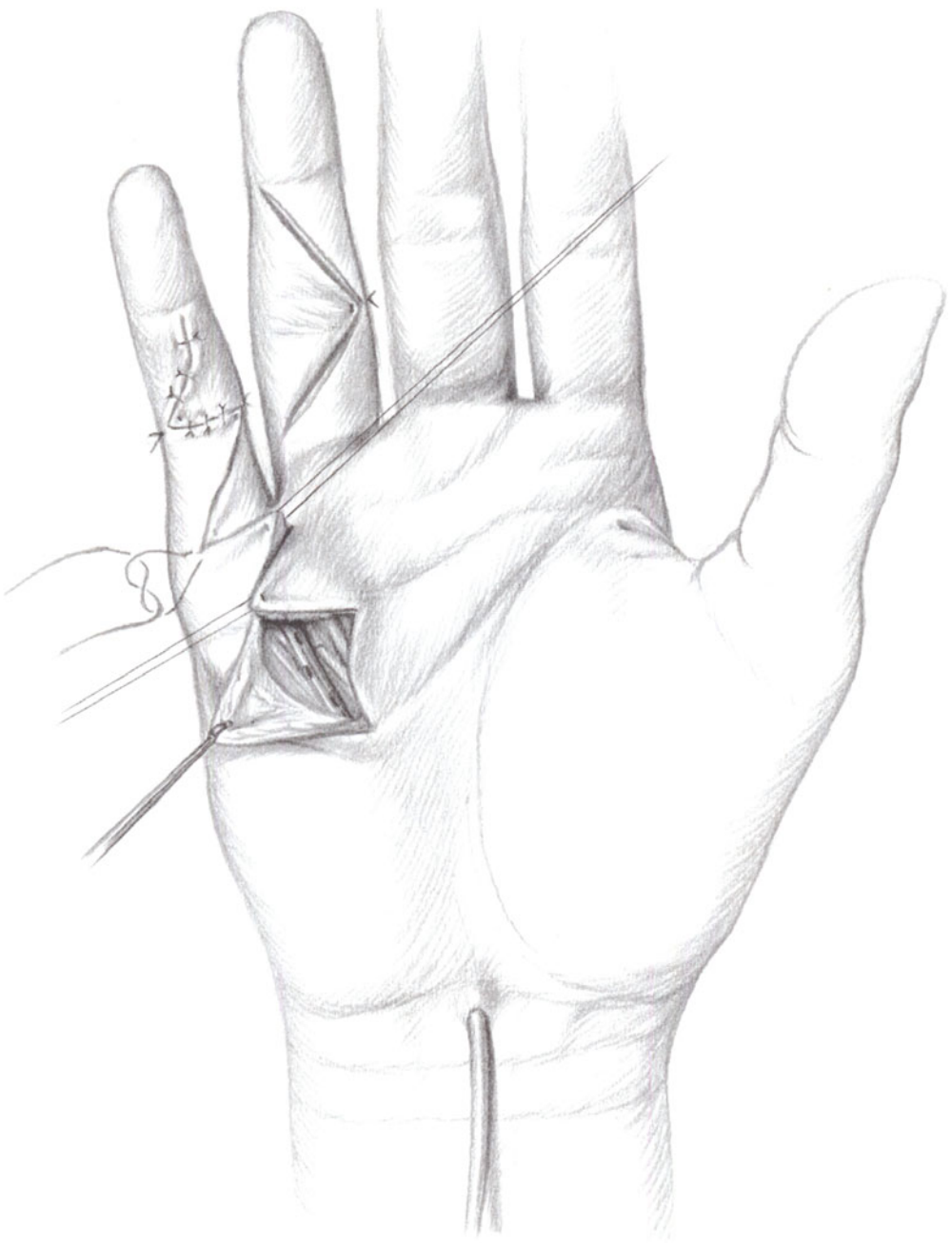


Fig. 44k. Planning and marking the 60-degree Z-plasties with rounded, broad flap tips to prevent necrosis of the apex. We place the Z-plasty into the digital flexion crease. After the tourniquet is released, the reactive hyperemia needs to subside. Meticulous hemostasis is achieved with bipolar cautery. Checkup for undisturbed flap perfusion. The flaps are resected and replaced by full thickness skin grafts or local flaps if the flap viability is questionable. A 10 Charrière suction drain is placed and secured. No buried (subcuticular) sutures should be used.

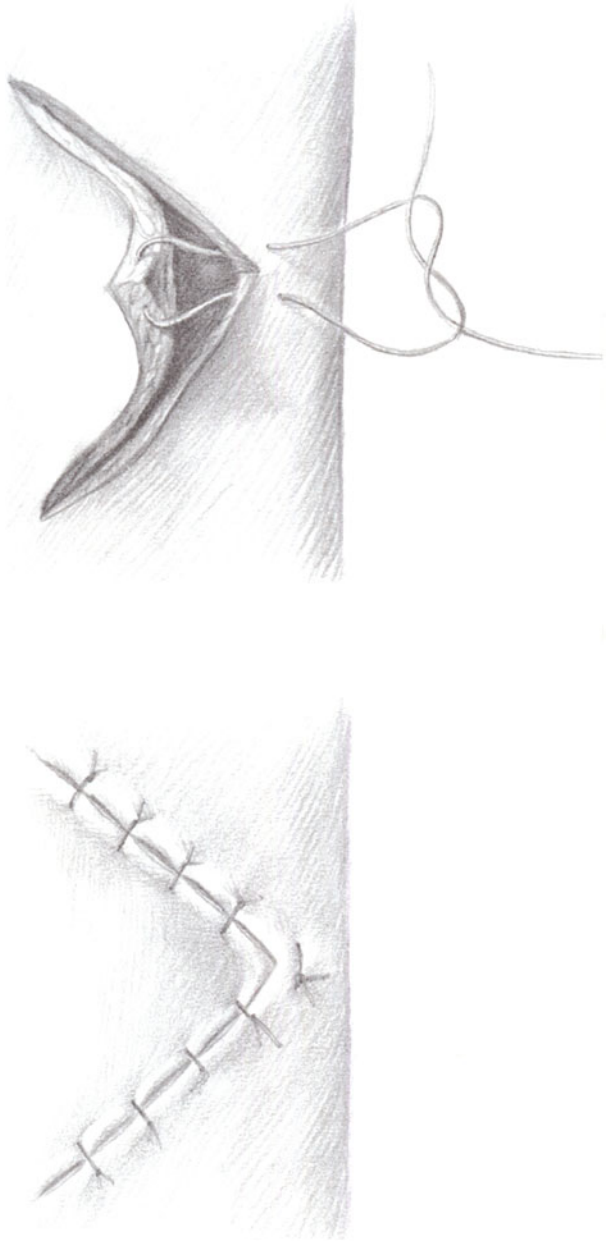


Fig. 44l. Interrupted skin sutures with 5-0 and intradermal Gillies corner-stitches at the flap tip to avoid apical necrosis.

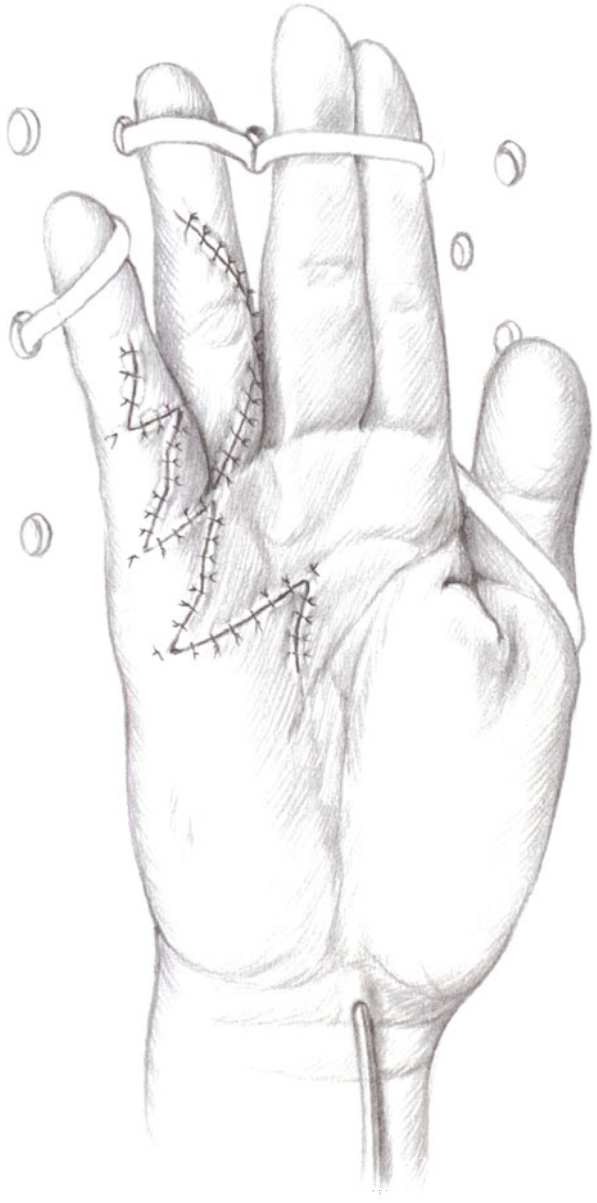


Fig. 44m. Finished line of sutures closing a Bruner incision on the ring finger.

proceeds close to the muscle belly of the opposite lumbrical muscle and exposes the corresponding neurovascular bundle. The dissection continues superficial to the flexor tendons taking care not to accidentally open the fibrous sheath of the finger flexor. The procedure continues on the ulnar aspect of the middle finger. The tenth neurovascular bundle on the small finger takes a separate course.

After the Z-flaps are created and after subtle hemostasis, the flaps are transposed and sutured. Details of wound closure with Z- and V-Y-flaps are given later.

20.3.5 The open palm technique

Originally introduced as a two-stage procedure, the open palm technique relies on only transverse incisions in the palm and at the level of the digits. These incisions are placed into the palmar or digital creases. For the one stage procedure, one can additionally use a lazy-S incision or a modified BRUNER approach. These techniques allow to proceed with a fasciectomy of the involved fingers and permit a primary closure of the longitudinal incisions, even in combination with the open palm technique (Fig. 45a).

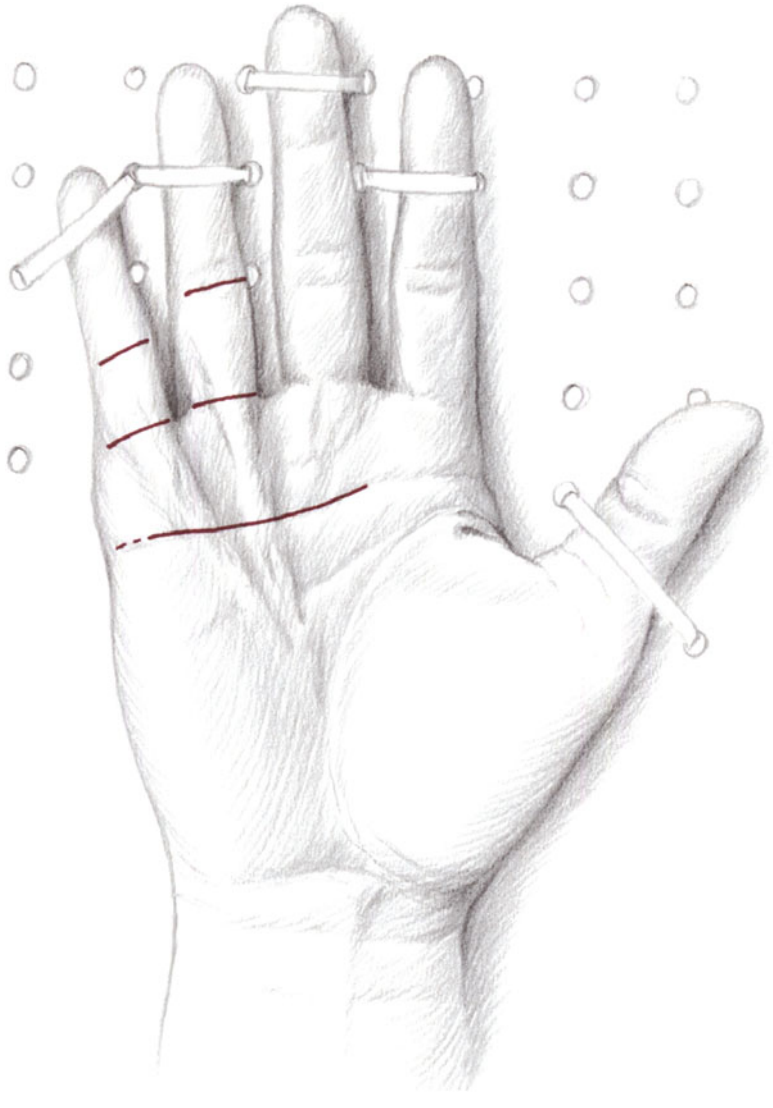


Fig. 45a. Open palm technique. Originally introduced as a two-stage procedure, the open palm technique always uses transverse incisions in the palm and on the fingers. These incisions are placed into the joint or palmar creases.

The partial fasciectomy is performed after the original McCash method (1964). However, it is limited to removal of the fascial tissue that causes the contracture and without any safety margins.

The digital dissection always ends at the proximal interphalangeal joints. Release of the distal interphalangeal joints necessitates a second operation, according to McCash (1964, 1986) and Lubahn (1966). The longitudinal digital incisions are closed, sometimes even an optional possible secondary palmar incision. The other transverse incisions located in the palmar creases remain open so that the puckered palmar skin can spread over the incisions and healing by secondary intention occurs (Lubahn, 1999; Fig. 45b–f).

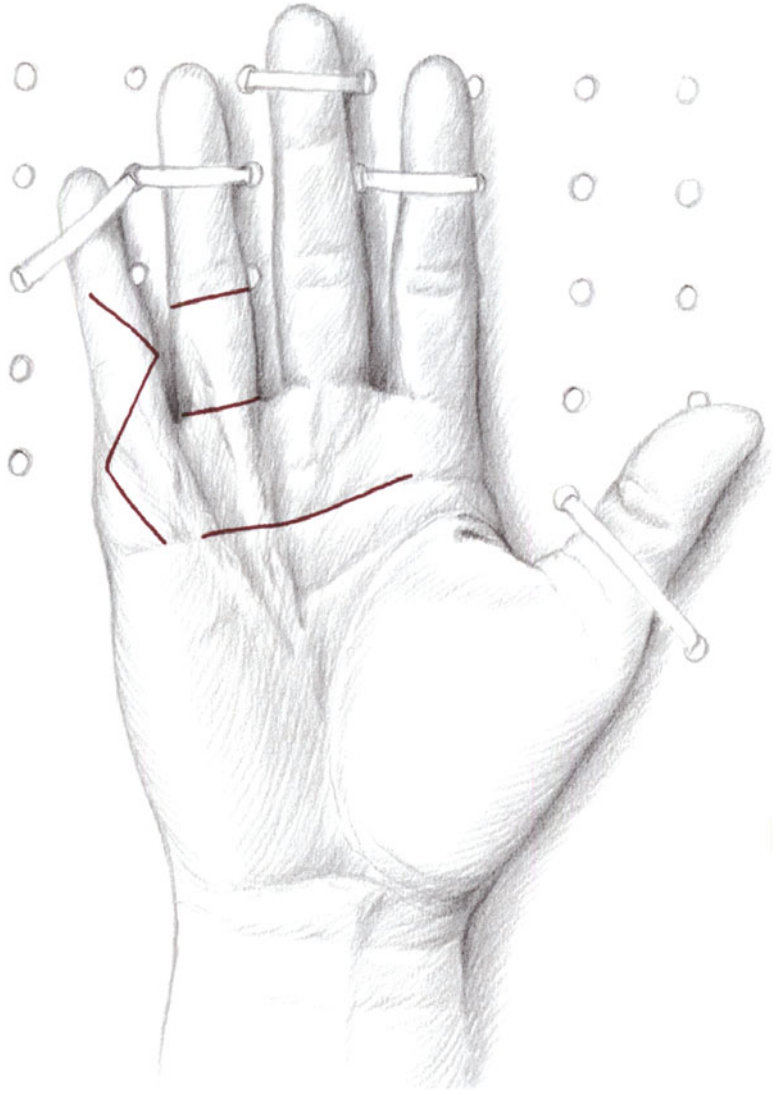


Fig. 45b. For the single-stage modification simultaneous fasciectomy at the affected fingers is an option. Serpentine or modified Bruner incisions are chosen , which allow primary closure of the longitudinal incisions even with the open palm technique.

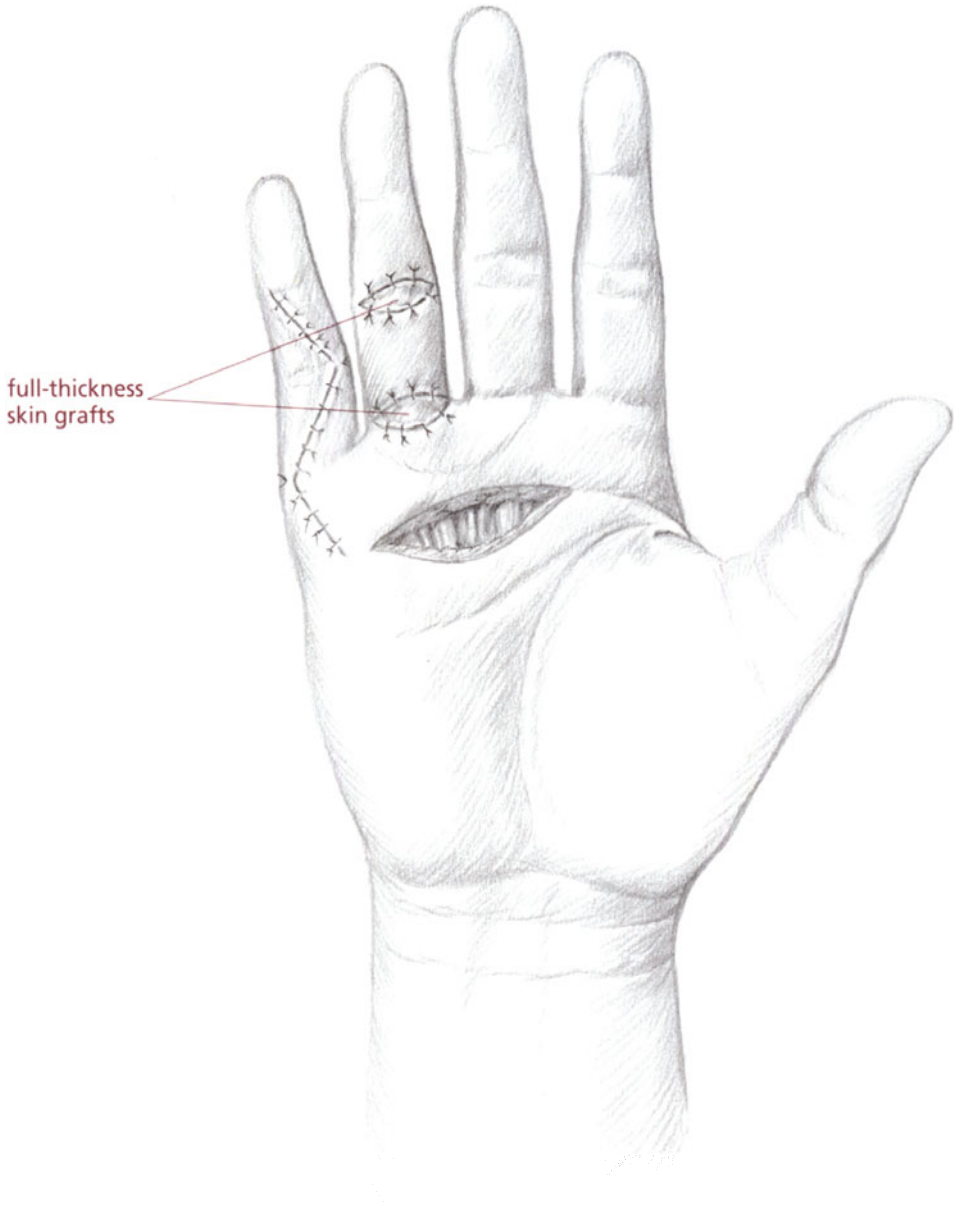


Fig. 45c. In the original McCash procedure, a partial fasciectomy is performed. But it is limited to the excision of the fascial tissue causing the contracture. No additional safety margin is kept. Please refer also to figure 44e. Dissection of the fingers always ends at the proximal interphalangeal joints. McCash and Lubahn point out that the release of the proximal interphalangeal joints always requires a second intervention. The longitudinal digital incisions and parts of the secondary palmar incisions are closed. If a primary closure of the transverse digital incisions cannot be achieved without tension, then full thickness skin grafts are obligatory if a well-perfused wound bed is present that can prove the necessary capillary outgrowth to vascularize the graft. The transverse crease in the hand remains open, permitting the palmar skin to spread during secondary wound healing within a few weeks.

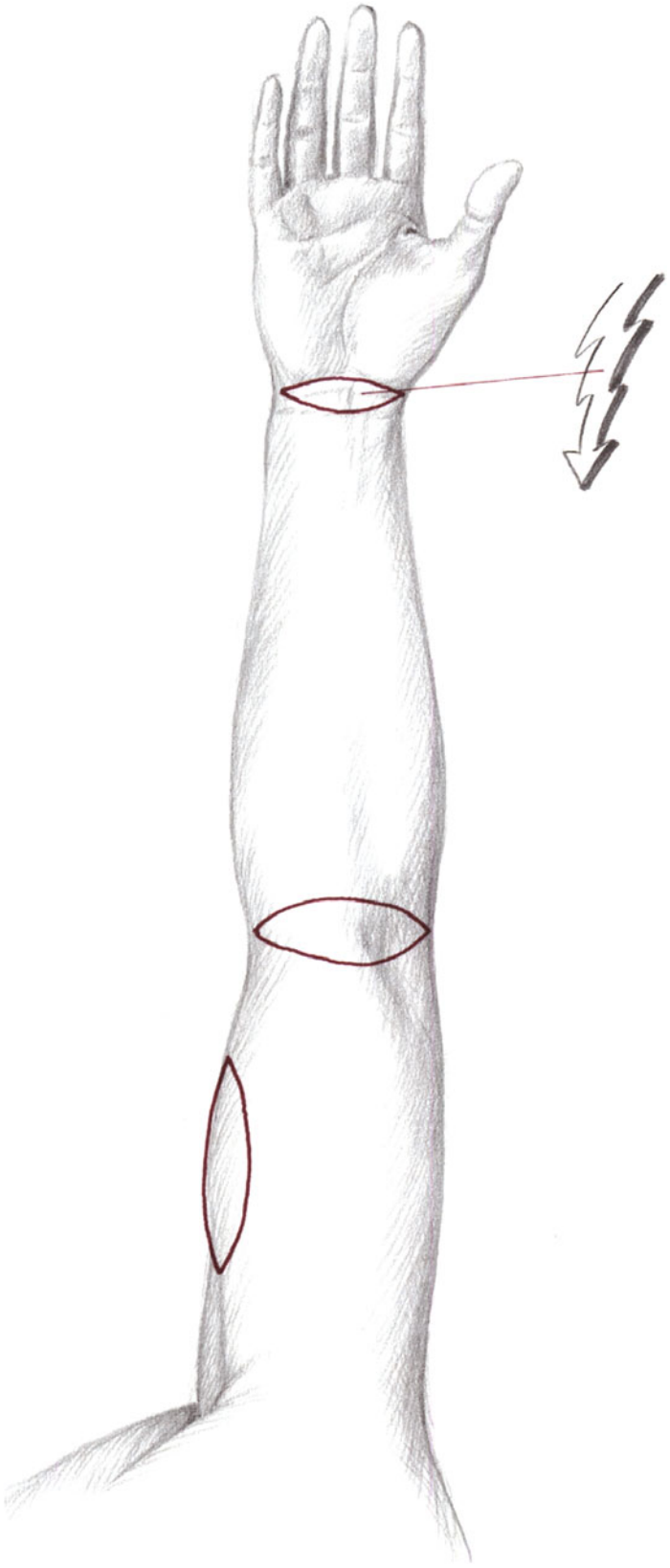


Fig. 45d. Donor sites for harvesting full thickness skin grafts. Basically the donor sites are always closed directly.

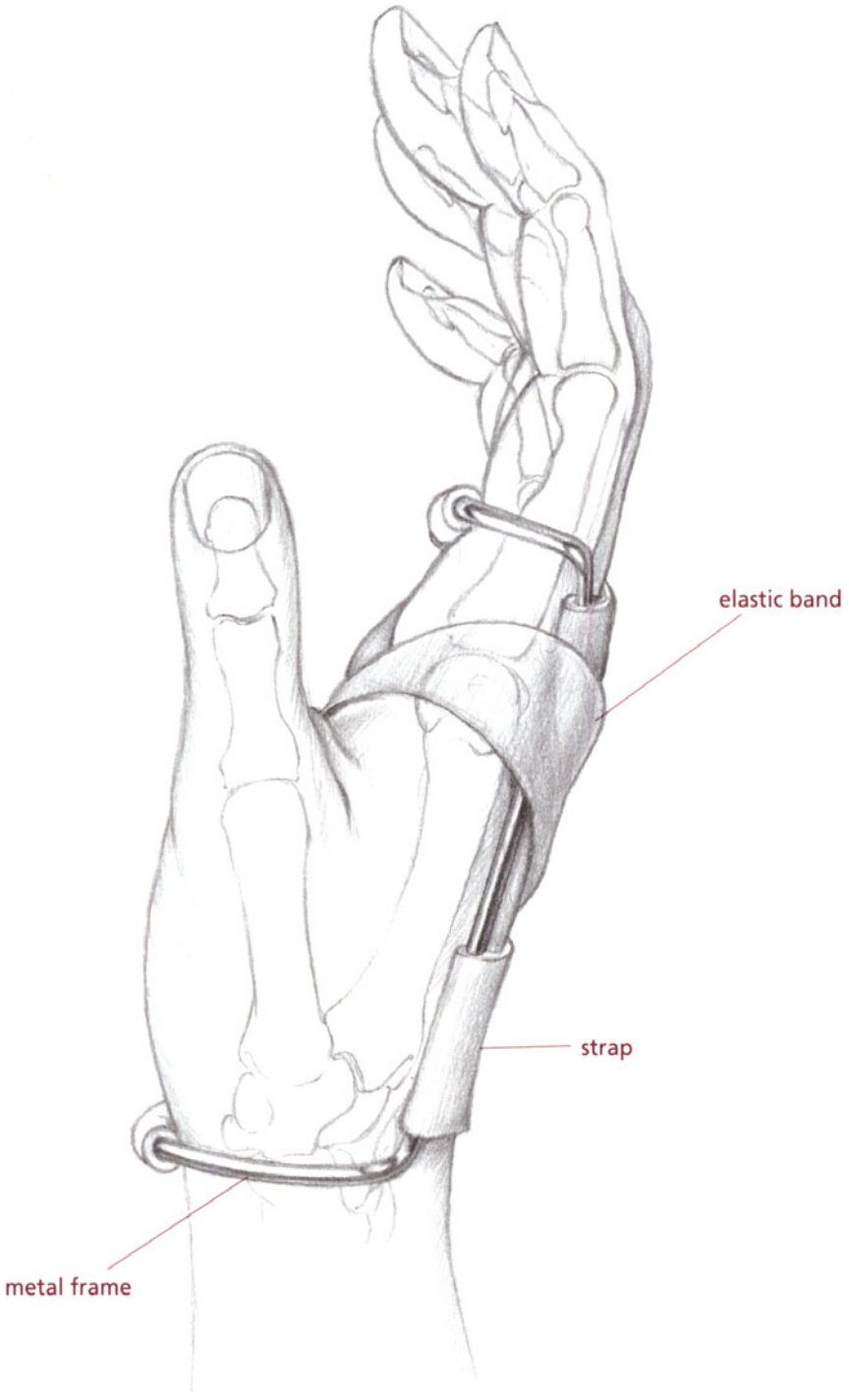


Fig. 45e. A strict rehabilitation program is an essential prerequisite for a successful functional outcome. This includes the consistent application of static night splints and dynamic extension splint during daytime. The picture shows a frame-shaped dynamic extension splint (made of metal) with a distal transverse elastic band and a proximal strap. Adjustable extension splint with frame support over the rasceta and metacarpophalangeal joints tightened by a dorsal strap.

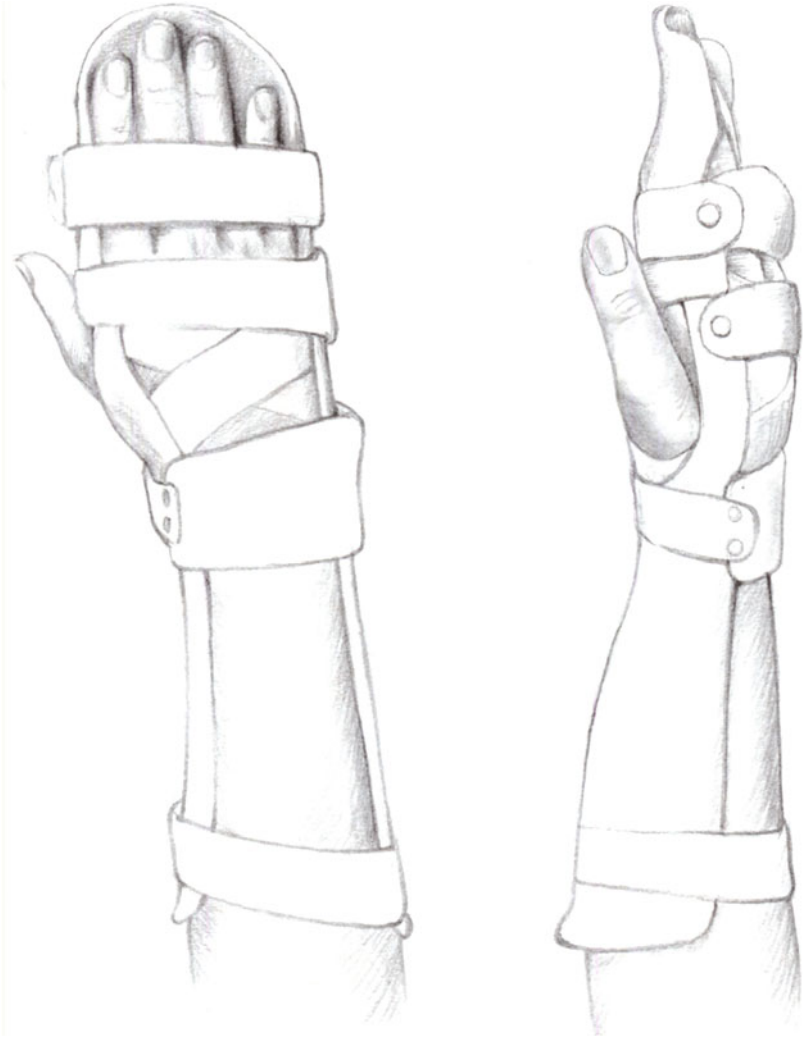
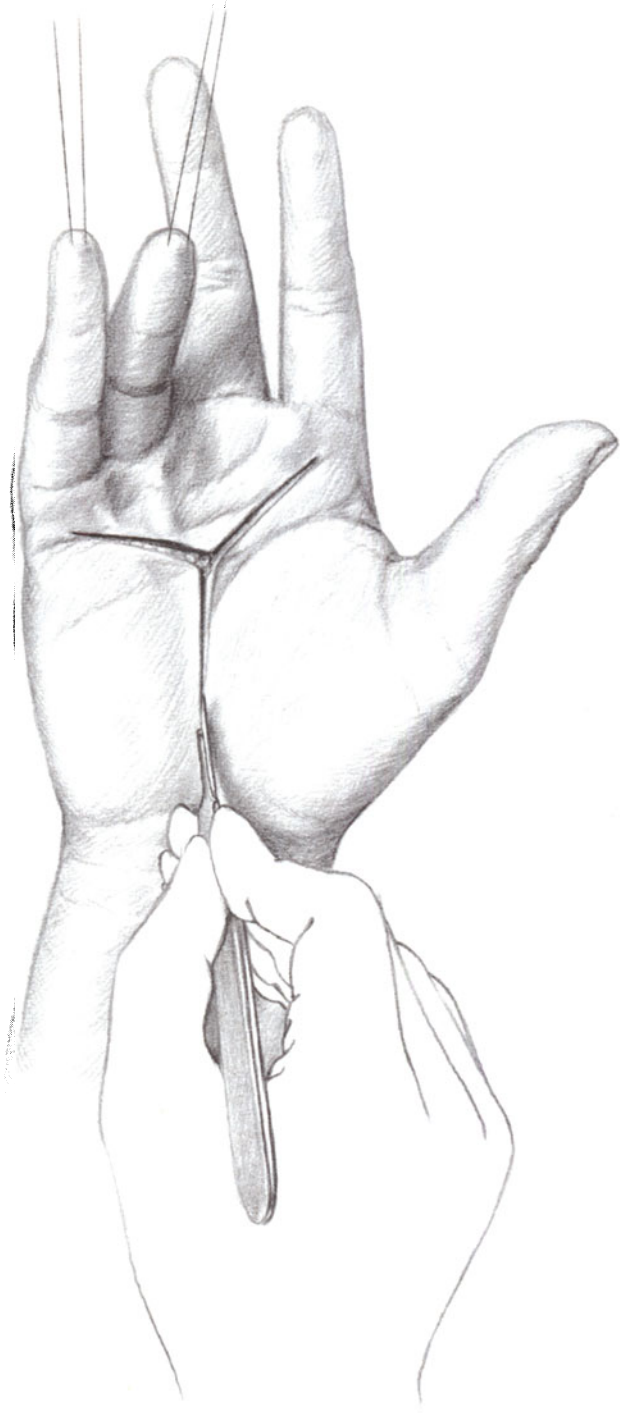


Fig. 45f. A static splint that holds the metacarpophalangeal and interphalangeal joints in full extension is worn at night.

20.3.6 Total fasciectomy

The palm is exposed through triradiate, or Mercedes star-like, skin incisions. The incisions are placed in the center of the palm, respecting the natural palmar and digital skin creases. The proximal incision runs along the middle longitudinal palmar crease and fuses with an "iatrogenic" transverse incision. The latter links parts of the proximal and the distal transverse palmar creases. A 120-degree angle between the limbs is optimal and is the best prevention against central skin necrosis (Figs. 46a).



The triangular flaps are undermined with the scalpel, which is not drawn but moved under application of a downward pressure. The broad flap bases guarantee adequate flap perfusion. Perforators from the monticuli, thenar, and hypothenar region nourish the flaps. These vessels should always be preserved to minimize wound complications (Fig. 46b).

MILLESI's approach provides excellent exposure of the entire diseased palmar aponeurosis to the apparently uninvolved zones. The central palmar fascia is dissected off the ulnar aponeurosis, the triangular fixation at the flexor retinaculum (insertion of the palmaris longus tendon) and the thenar eminence. The superficial palmar arch as well as the motor branch of the median nerve is always prone to injuries. Prograde dissection proceeds on top of the neurovascular bundle from proximal to distal and from radial to ulnar. The vertical septa are excised exactly as described for the partial fasciectomy (Fig. 46c).

If additional dissection of the digital fascias is required, small skin bridges remain between the digits and the palm, preserving also the monticuli. Possible longitudinal incisions are interrupted by Z-plasties, as previously described.

After releasing the tourniquet, the wound is temporarily packed with warm saline sponges until reactive hyperemia has settled. After their removal, meticulous hemostasis follows and suction drains are placed. The skin is sutured; vaselined gauze dressings plus a compressive dressing of autoclaved steel wool are applied. A plaster splint is placed (Fig. 46d–f).

Fig. 46a. Total fasciectomy: Triradiate, y-shaped incision in the hollow of the palm, utilizing the natural creases. The dissection does not extend beyond the triangular palmar aponeurosis and ends at the thenar and hypothenar eminences and the monticuli, to safeguard perfusion. 120-degree flap wedges are chosen to prevent central necrosis.

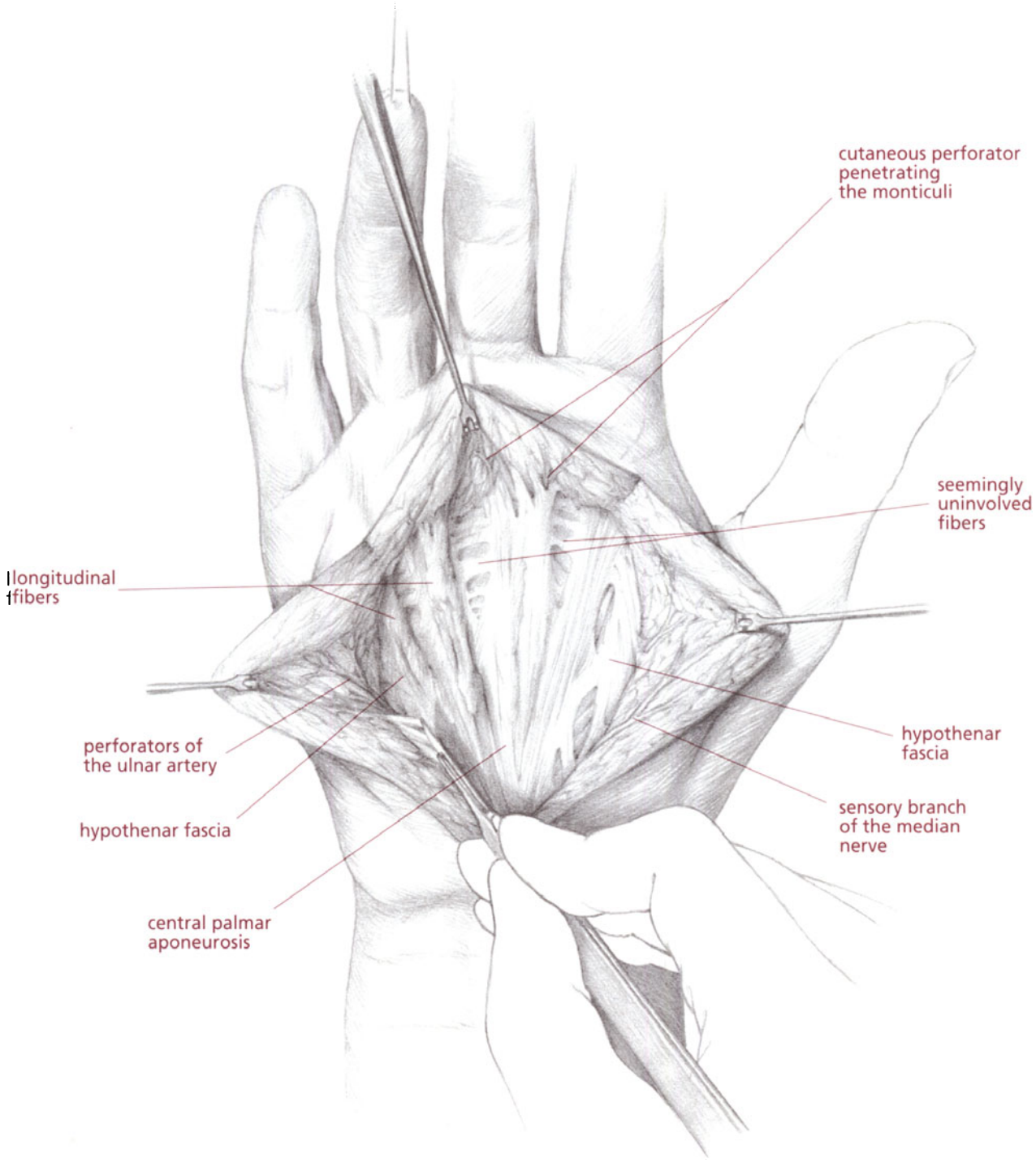


Fig. 46b. Sharp dissection of the triangular flaps with the scalpel. Be careful to keep the broad flap base and the subdermal plexus intact.

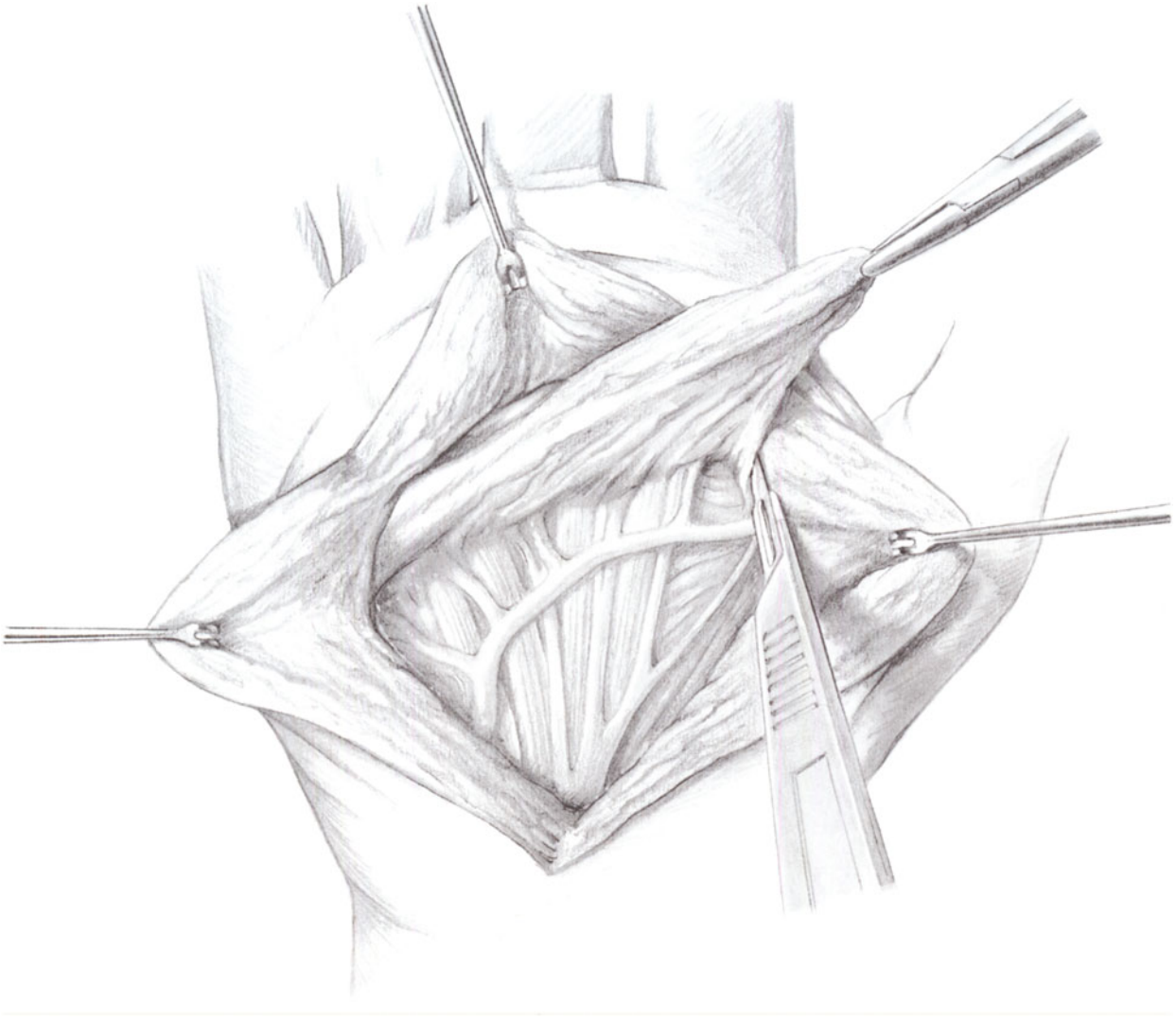


Fig. 46c. Exposure of the entire central palmar aponeurosis, including healthy and fibromatous parts. The palmar fascia is detached from hypothenar eminence with small chopping movements; also from the triangular attachment at the flexor retinaculum (palmaris longus insertion) and from the thenar eminence. The superficial palmar arch and motor branch of the median nerve are particularly prone to injury. Prograde dissection proceeds superficially to the visualized neurovascular bundle, from proximal to distal and from radial to ulnar. Vertical septa are also resected, as already described under partial fasciectomy.

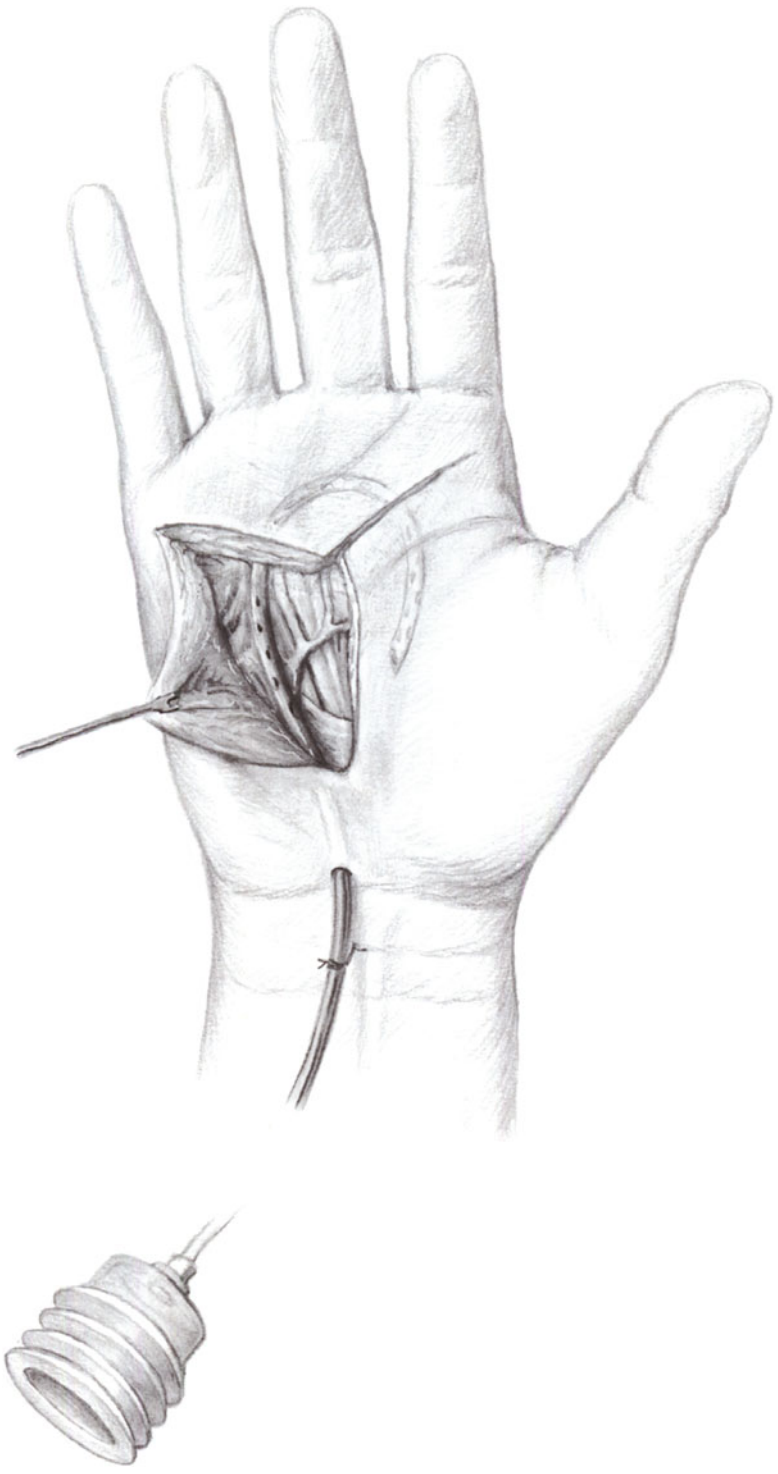
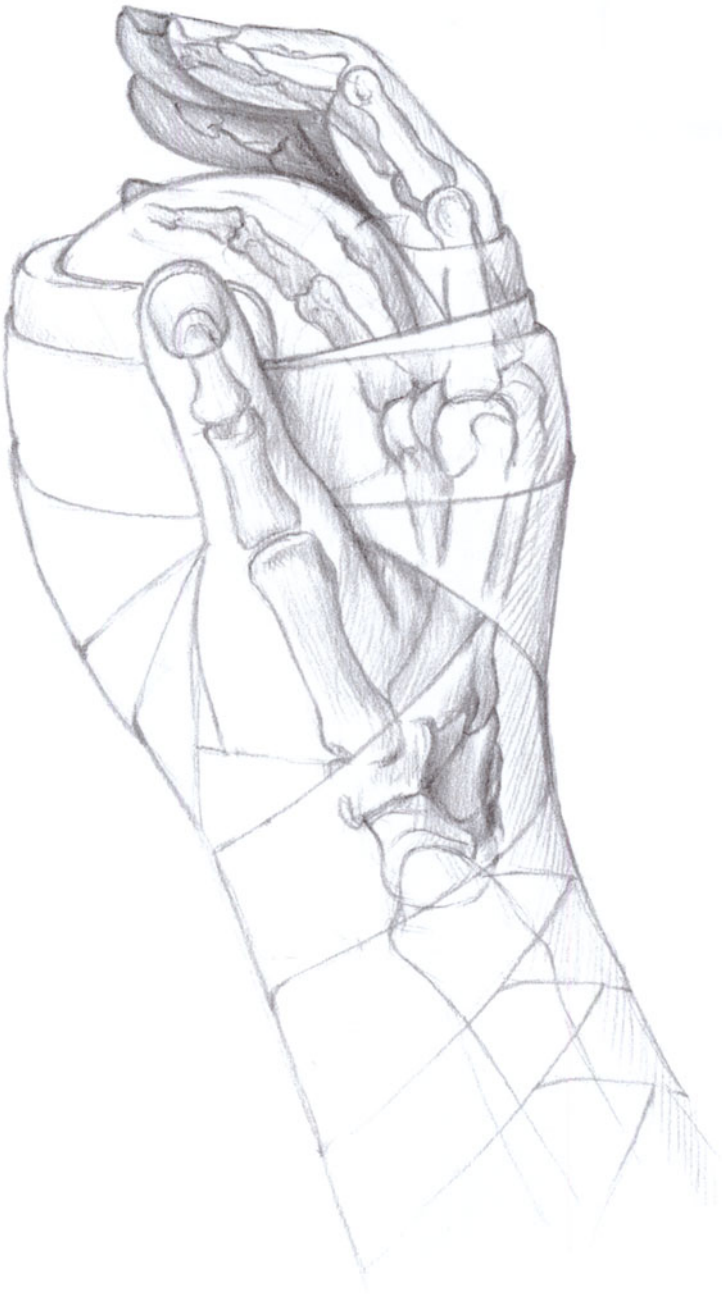
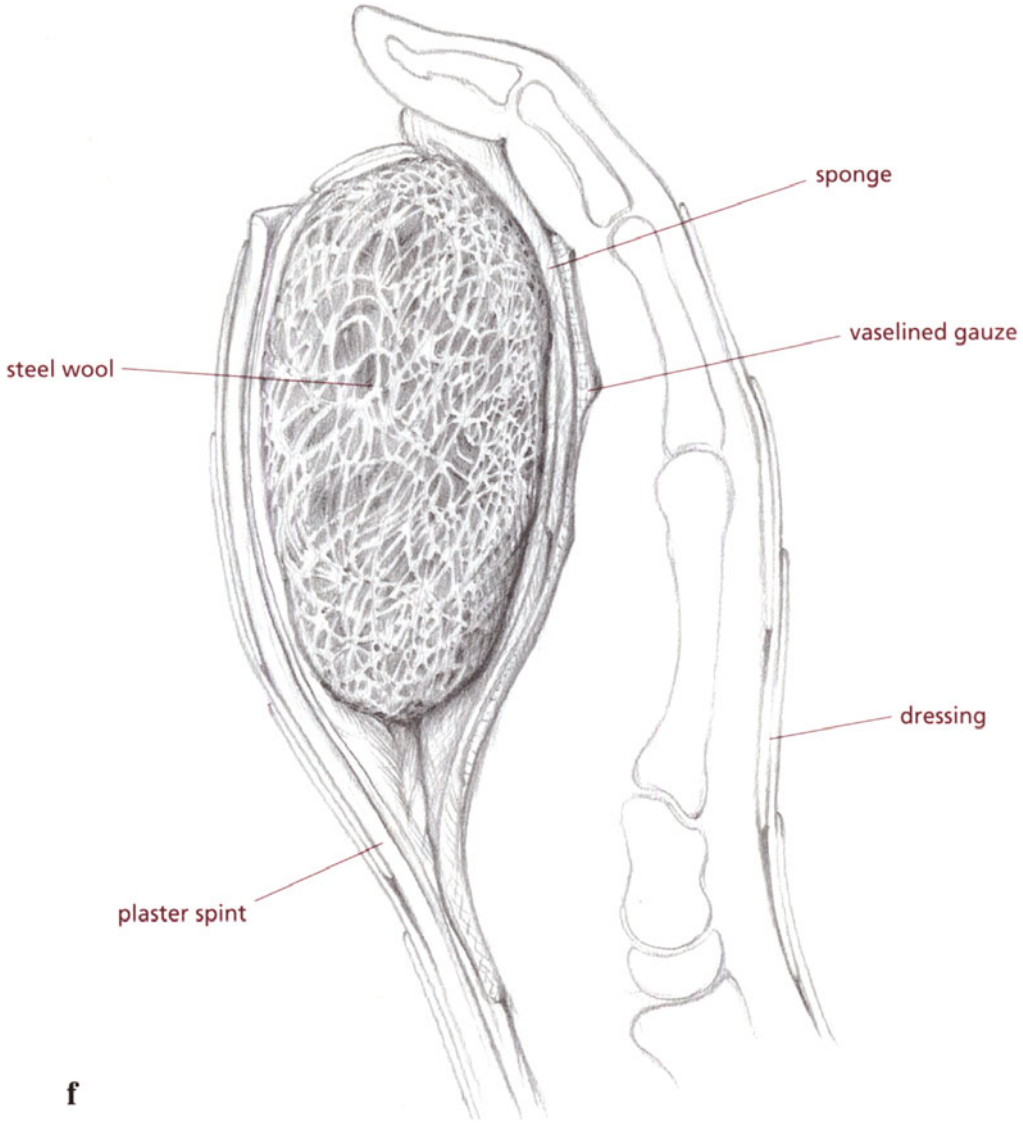


Fig. 46d. Completed total fasciectomy of the palm. If additional digital dissections are required, small skin bridges remain at the level of the digitopalmar transition zone. The digital longitudinal incisions are interrupted by Z-flaps, as shown under partial fasciectomy. After the reactive hyperemia has subsided and after careful hemostasis, a suction drain is placed.



e

Fig. 46e, f. Skin suture, vaselined gauze dressing, compression bandage with autoclaved steel wool and plaster splint. The very short time of immobilization makes it possible to dispense with the mandatory intrinsic-plus position in favor of a functional position, without any disadvantages for the collateral and capsular ligaments. When no grafts were used, we remove the dressing and plaster splint already on the fifth postoperative day. Intense active physiotherapy of the operated hand is obligatory.



f

20.3.7 Dermofasciectomy

In dermatofasciectomy, a radical local resection of both the fibrotic skin and digital fascias is performed. The resection extends from the distal transverse palmar crease to the distal palmar interphalangeal crease and laterally to the midlateral line, while the flexor tendon sheath as well as the bilateral neurovascular bundles are preserved. Beyond the skin affected by Dupuytren's disease, the safety margin is 5 mm (Fig. 47a). As a prophylactic measure against contracture, it is advisable to use a zigzag or wavy excision beyond the midaxial line at the joint creases. These indentations help to fit the graft into the resulting compound defect and facilitate suturing (Fig. 47b). An appropriately shaped graft is harvested.

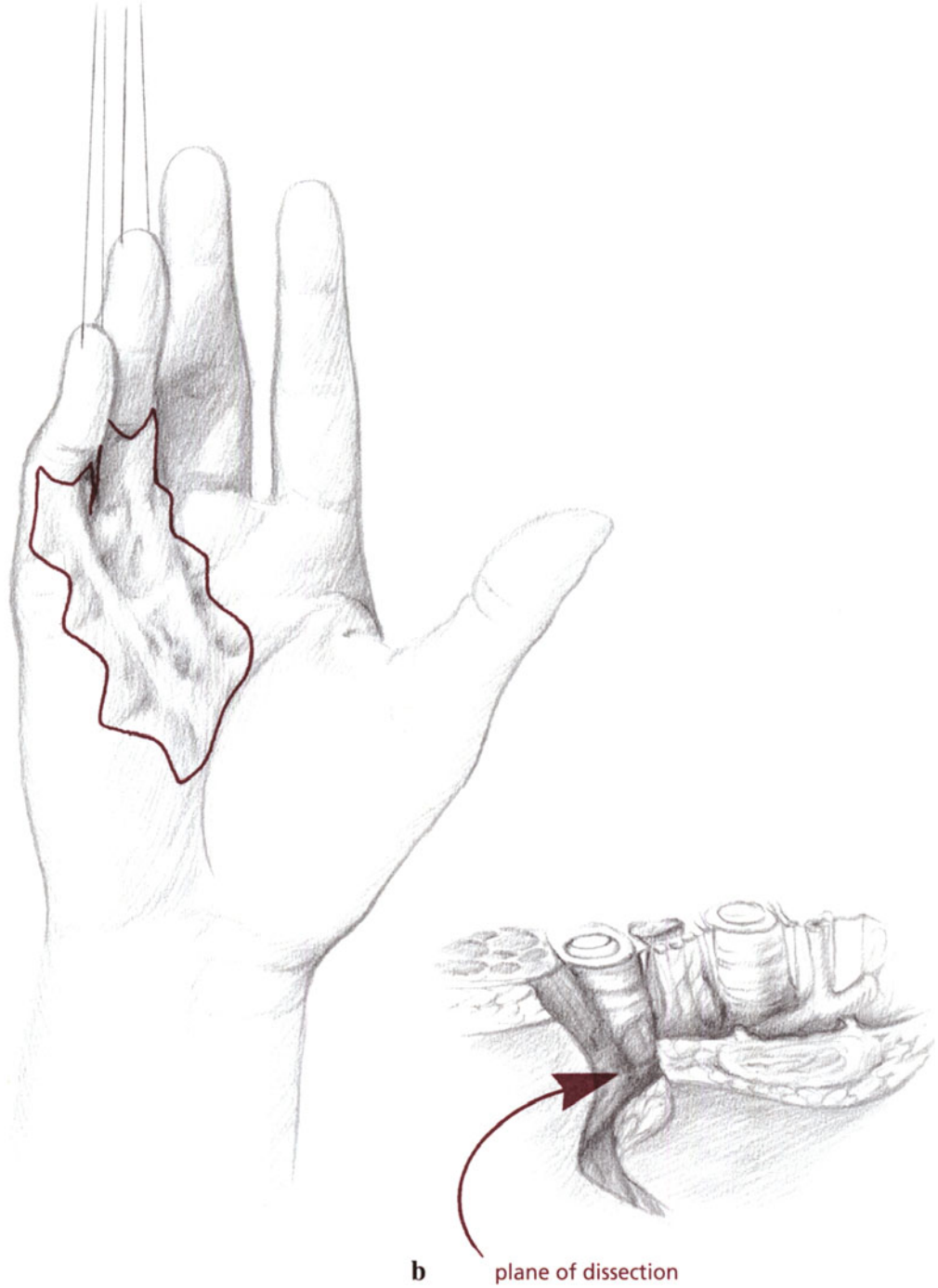


Fig. 47a and b. During dermatofasciectomy, the fibrotic area is resected en bloc with a safety margin (**a**). **b.** The palmodigital level of dissection is superficial to the neurovascular bundles while preserving the flexor tendon sheath to maintain a suitable recipient site for grafts.

The defect is covered with a full thickness skin graft, which is harvested from the flexor side of forearm or the medial upper arm (BROTHERSTON et al., 1994 (Fig. 47c, d).

Prior to applying sutures, the tourniquet is released and hemostasis achieved with bipolar cautery. Simple holes are cut into the graft to avoid fluid collections. A tie-over pack is applied, supplemented by a plaster splint.

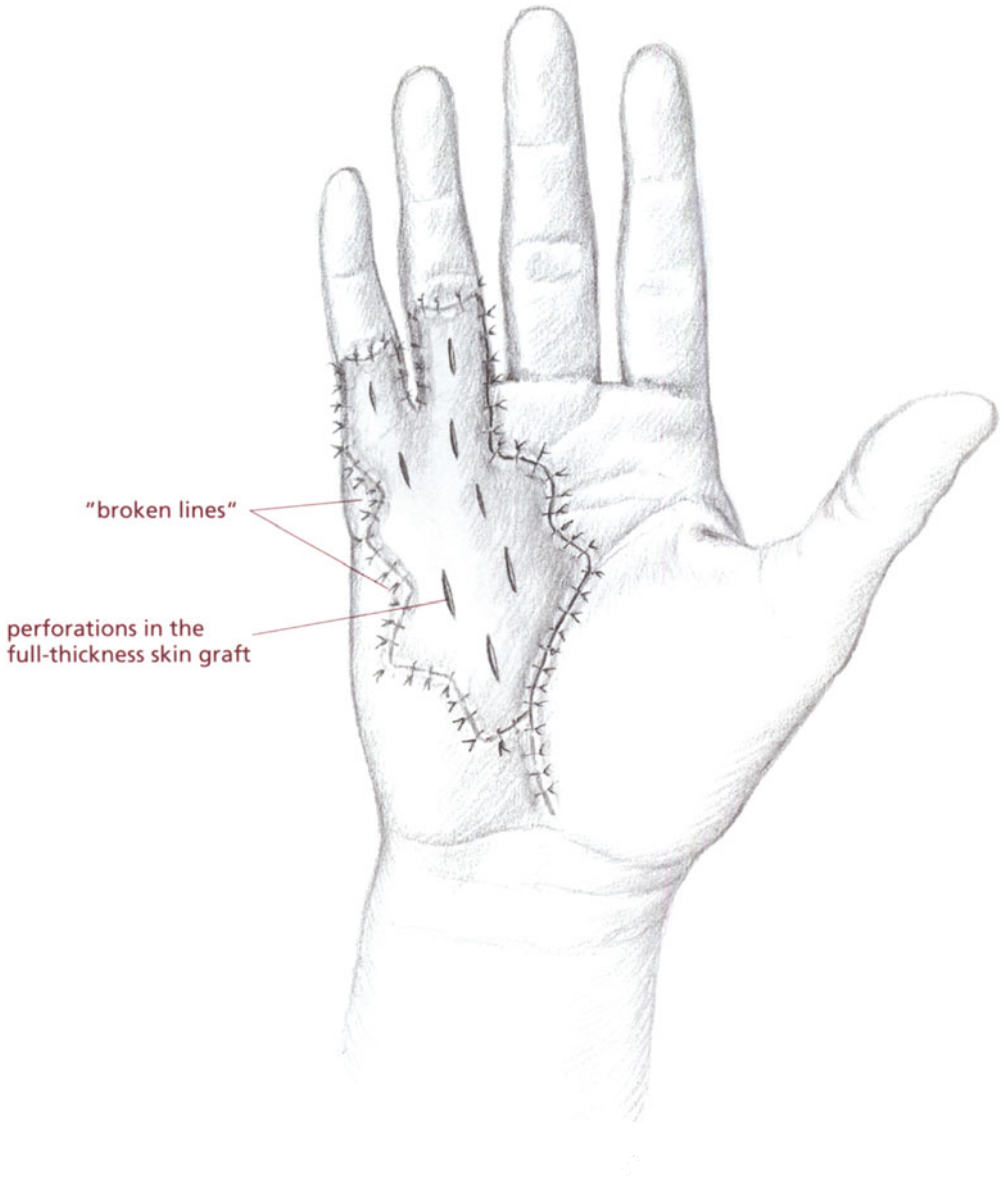


Fig. 47c. Because of scar shrinkage and cicatrix contracture, it is important that the borders of full thickness grafts are not straight, but angulated, curved or interrupted by bridges of intact skin. Multiple iatrogenic perforations of the skin graft (scarification) drain hematomas or other fluid collections underneath the graft. The grafts are secured with a tie-over dressing. A plaster splint provides immobilization.

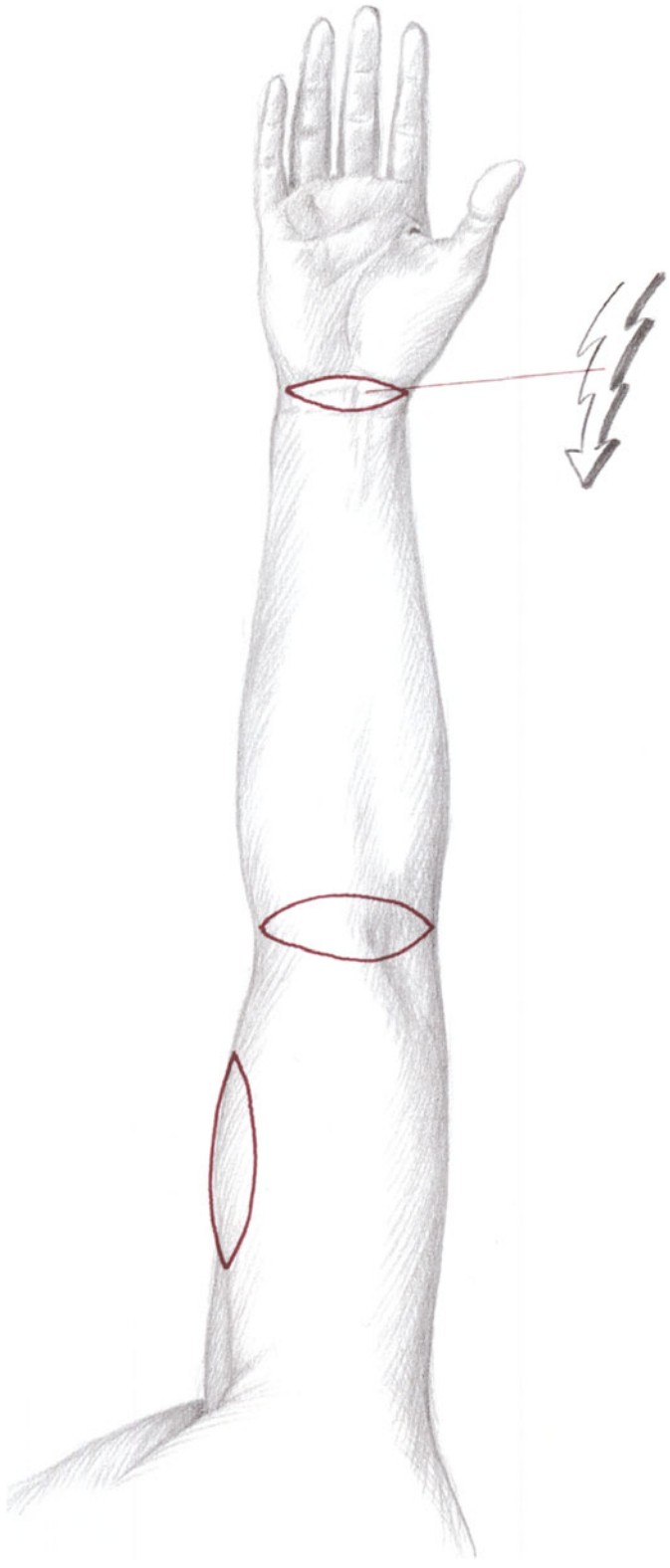


Fig. 47d. Possible donor sites for spindle-shaped full thickness skin grafts on the ipsilateral limb are the medial aspect of the upper arm, the antecubital fossa, and, for smaller grafts, the rasceta. The necessary graft size is determined by a template of the defect. All donor site areas are closed primarily. In singular cases of expanding defects even multilocular donor site areas for skin grafts are necessary to achieve a tension-free, complete and one-stage resurfacing. The donor site near the rasceta has two disadvantages: (1) The scar tends to hypertrophic or keloid formation and may hinder the patient from wearing a watch or jewellery. (2) A visible scar on the proximal,or distal wrist crease may be thought to be secondary to a suicide attempt.

20.3.8 The continuous elongation technique

For the continuous elongation technique (Messina, 1994), two self-tapping pins with a diameter of 2–3.5 mm are inserted into the ulnar side of the fourth and fifth metacarpal, making sure to include of all four cortical layers. Simultaneously a transverse Kirschner wire is placed in the proximal phalanx of the middle, ring or little finger. This wire will later be bent to form a traction loop. The T.E.C. device ("Tecnica di Estensione Continua"), the current model of which weighs 190 g, is attached to these steel parts. These measures should only be performed on inpatients. After the third postoperative day, aftercare can be provided on an outpatient basis. During the first three to four days, daily painless lengthening of 3–4 mm can be achieved. Over the following three weeks, lengthening is augmented by progressively twisting a turnbuckle, which translates traction into extension of the formerly flexed fingers. The accomplished favorable effects on the metacarpophalangeal and proximal interphalangeal joints can be a preparatory step for conventional fasciectomy. The use of this technique is especially advantageous for surgeons not experienced in plastic surgery, or in situations, where one wants to avoid grafts or flaps (Messina and Messina, 1991; Messina, 1994; Fig. 48a, b).

The inaugurator of this method obtained "good results" in 30 patients. The treatment was not aborted in any cases. No infections or other negative aspects were reported.

Despite the apparently excellent results, Hodgkinson introduced an advanced, miniature model of the device already in 1994 (Pipster).

Citron and Messina (1998) responded with the development of a similar device, which resembles the Pipster, and named it the Verona apparatus. It was applied just like the Pipster. The authors recommend primary use of the miniature Verona fixator in fixed PIP flexion contractures.

Because of the obvious disadvantages of the Messina procedure, such as possible nerve and tendon injuries while drilling, pin tract infection, poor patient acceptance of the permanent "external fixateur" (T.E.C. device), and because of the high costs of this treatment, Piza-Katzer et al. (2000) introduced a non-invasive, intermittent pneumatic balloon extension procedure for the contracted fingers. For this purpose a metal plate is custom fitted to the palm on the basis of an imprint. Distally, the plate extends to the metacarpophalangeal joints. This metal plate serves as a stable abutment and also secures the balloon. An assembly similar to a metacarpal orthesis holds the pneumatic dynamic extension splint with a Velcro fastener. The balloon pressure is 0.35 bar. The patient increases the pressure slowly with the help of an ordinary bicycle pump. The extension needs to be painless, however. Pain signifies that the end point of the currently achievable pliability has been reached. Piza-Katzer et al. (2000) express that this procedure permits a degree of extension, which contributes significantly to the reduction of complications after a subsequent fasciectomy. Another advantage of this method is the fact that bent digits can be much easier washed and otherwise cared for (Fig. 48c).

Fig. 48a. Dupuytren's disease of right middle, ring and little fingers. The latter has a flexion contracture of 160 degrees.

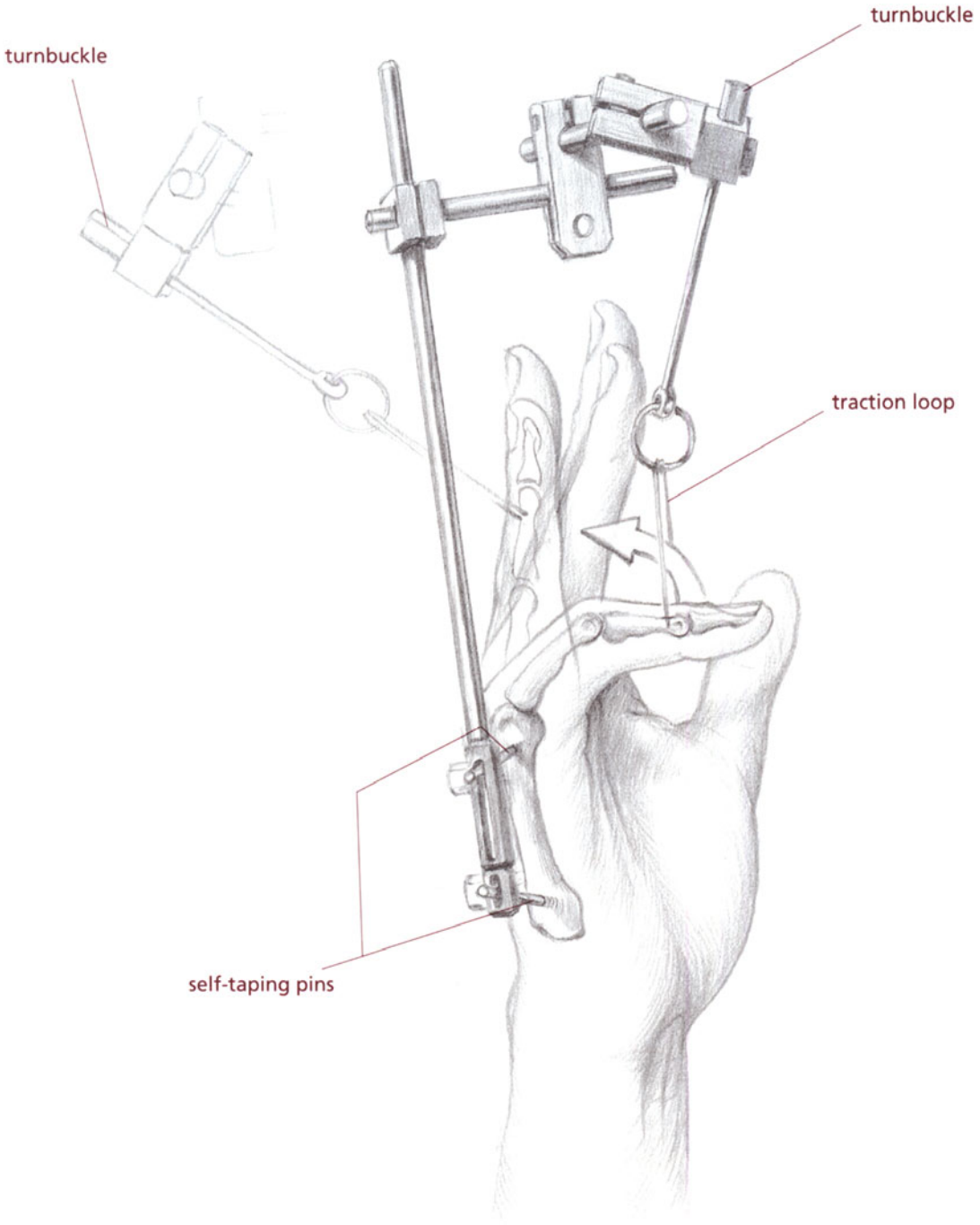


Fig. 48b. Messina's T.E.C apparatus (Biotek, Turin) for continuous extension treatment in Dupuytren's disease. The end jaws of the longitudinal bar (smooth) hold two self-tapping Steinmann pins (2–3.5mm diameter) in place. The second jaw holds the grooved cross bar in place, which permits free length adjustment. The third jaw, which is fixed to the cross bar, permits the height to be adjusted according to degree of contracture. The other jaws on the smooth cross bar can be changed in terms of abduction. The entire apparatus aims to straighten the afflicted little finger through continuous extension (elongation) via a traction loop that is fixed at the middle phalanx. The original T.E.C. apparatus weighs 190 grams.

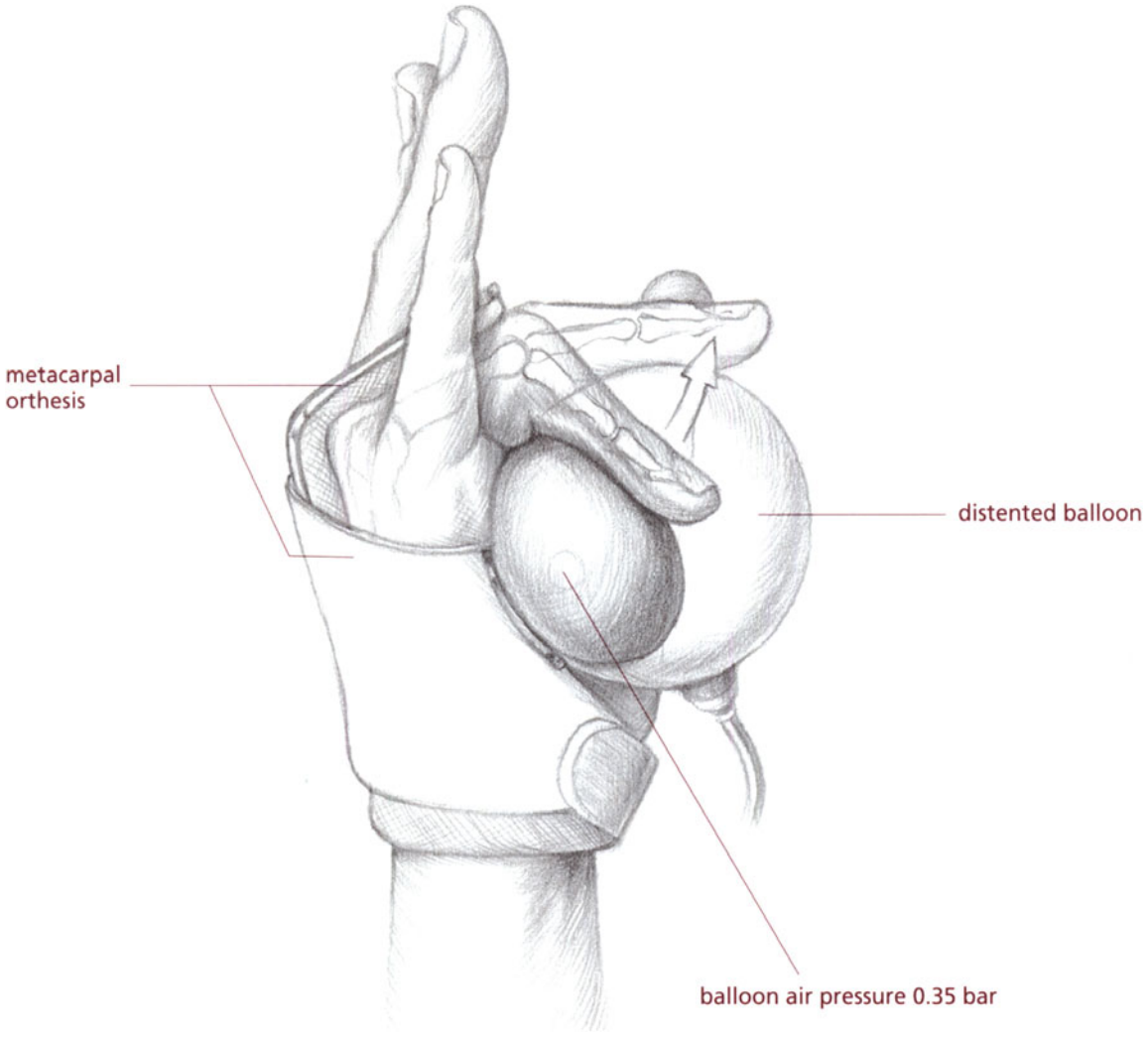


Fig. 48c. Pneumatic balloon extension: On the one hand, the stable metacarpal orthesis serves to attach the "balloon" used for extension treatment, on the other, it provides intermediate fixation of the proximal phalanx to transmit the counter-force. Initial balloon air pressure 0.35 bar. With a commercial bicycle pump, the pressure is increased until the pain threshold is reached. The pneumatic extension device aims to achieve full finger extension over a prolonged period. The arrow indicates the direction of digital extension (elongation).

20.4 Skin suture

Unfortunately, primary wound closure can be accomplished in only few patients with Dupuytren's disease. If the recipient area does not meet the requirements for a skin graft, a variety of flap options can be considered. The easiest and most reliable flap will always be considered first. The selection progresses from the easiest to the most difficult.

Only the most common of the various flap types are introduced here; we are not aiming to be comprehensive.

20.4.1 Skin grafts

Coverage with skin grafts always requires a well-perfused recipient area. Full thickness skin grafts are the primary choice, because they are very sturdy and do not shrink much (BRENNER, 1998). The most suitable areas for harvesting full thickness grafts are the medial aspects of the upper arms, the antecutibal fossa, and both forearms. When used as a skin graft donor site, the rasceta is prone to hypertrophic scar formation. Scars in this area may also be stigmatizing, as they may be thought to have resulted from a suicide attempt. Common remote donor sites for skin grafts include the groin or the supraclavicular fossa. Grafts should not be harvested near the clavicles, because incisions in this region are likely to form hypertrophic scars or keloids (Fig. 45d; Fig. 47d).

20.4.2 Homodigital flaps

Zigzag incisions as recommended by BRUNER are preferably used in moderate contractures caused by Dupuytren's disease.

In low-grade contractures of up to 45 degrees, the perpendicular distance from the first flexion crease to the second digital flexion crease is 7–10 mm. Because of the fibromatous puckering of the skin, this will appear too short (HURST, 1996).

After resection of the afflicted cord, the aforementioned perpendicular distance in full extension of the finger will exceed 15 mm. The apparent skin shortages require either V-Y-advancement of the BRUNER flaps or skin grafting of the residual defects. Alternatively, the residual defects can be left open to avoid any strangulation of the digit (BENDISCHI, 1990; BENSON et al., 1998; Fig. 49a–e). The fourth option is an additional Z-plasty of the diagonal palmar incision.

The Z-flaps are planned and created at the end of the operation, when it is obvious which areas may be poorly perfused.

The common inclination angle is 60 degrees; more length is gained with greater angles. Large Z-plasties may create ring constrictions, however. Reduction of the angle of the triangular flaps simplifies transposition, but may be disastrous for the blood supply (< 45 degree) and result in necrosis. It is vital that the flap contains as much subcutaneous fat as possible (BRENNER, 1989).

If the flaps are not immediately created in alignment with the interphalangeal joint creases, it may be helpful to first incise the Z-flap unilaterally, before the corresponding side of the Z is determined on the opposite side. This is done by transposing the first triangular flap by traction with a Gillies hook to its corresponding point on the opposite side, in order to delineate the contralateral Z side. Then the second triangular Z-flap is cut. Careful hemostasis is performed with bipolar cautery. If the flaps' viability is questionable, they have to be resected and replaced by full thickness skin grafts or other local flaps. If the flaps are well perfused, tension-free, interrupted 5-0 sutures will complete the procedure. At the flap's tip, the Gillies corner stitch technique should be used to avoid necrosis.

If it is better to place the Z-flap directly in the flexion creases of the fingers or midway between them is still controversial. MCGROUTHER (1999) confirms that no scientifically valid, definitive conclusion has been reached. He also warns that the changing length-to-width-ratio may give rise to constriction rings when the flaps are placed in the flexion creases.

If the flexor tendon sheath has been opened accidentally, SKOOG (1974) recommends resurfacing the denuded tendon sheath with Z-plasties (mobilized triangular, palmar transposition flaps). The donor area can be resurfaced with skin graft (Fig. 50a–g).

Technically simple, laterodigital transposition flaps (BUNNELL 1956) are random pattern flaps. They are tongue-shaped flaps, which have a proximal base and stem from the lateral side of the finger. They are elevated above the neurovascular bundle, in order to close, for example, palmar defects caused by extending the contracted finger. The donor areas of laterodigital flaps are always covered with skin grafts (Fig. 51a–51c).

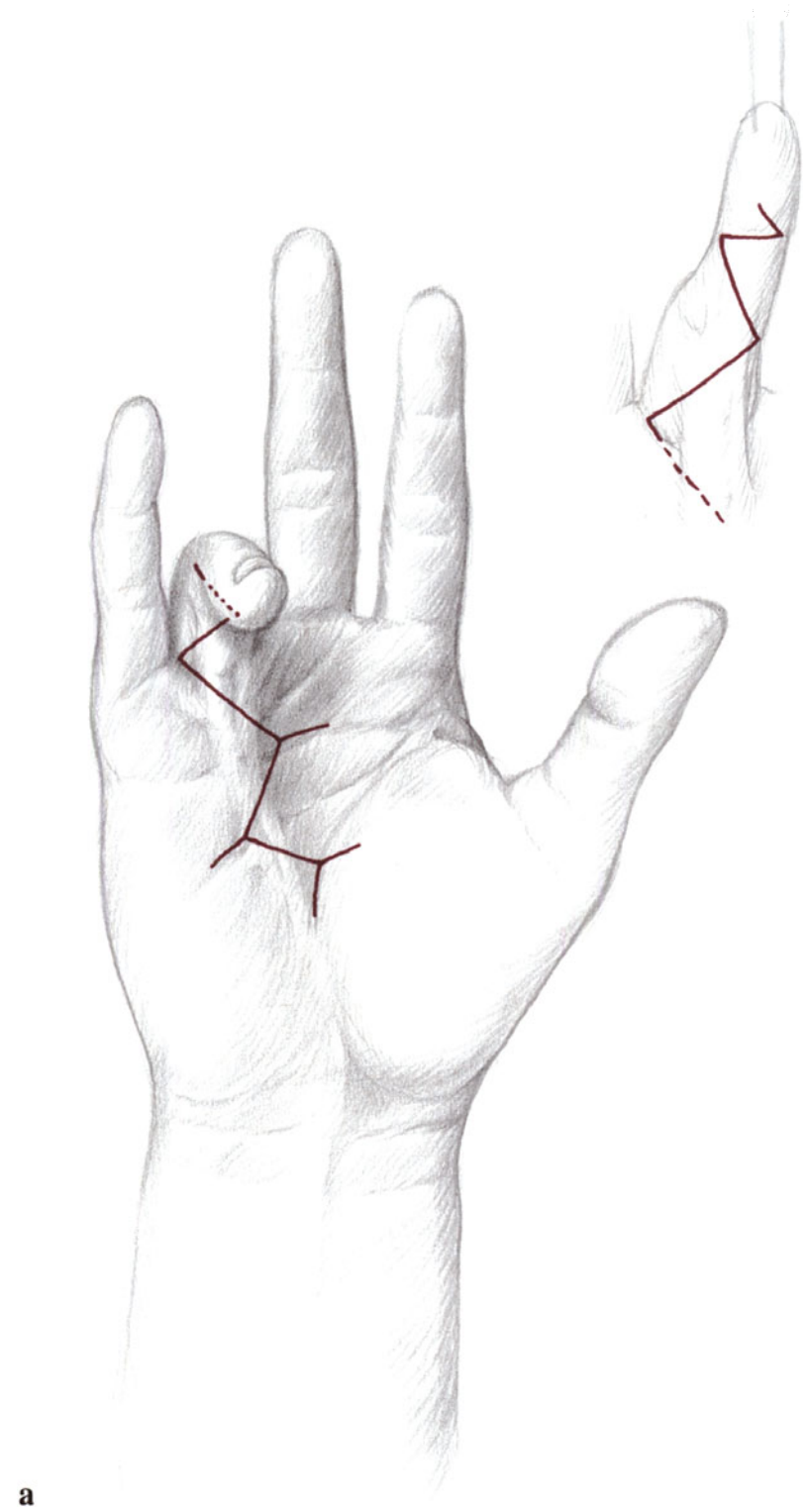
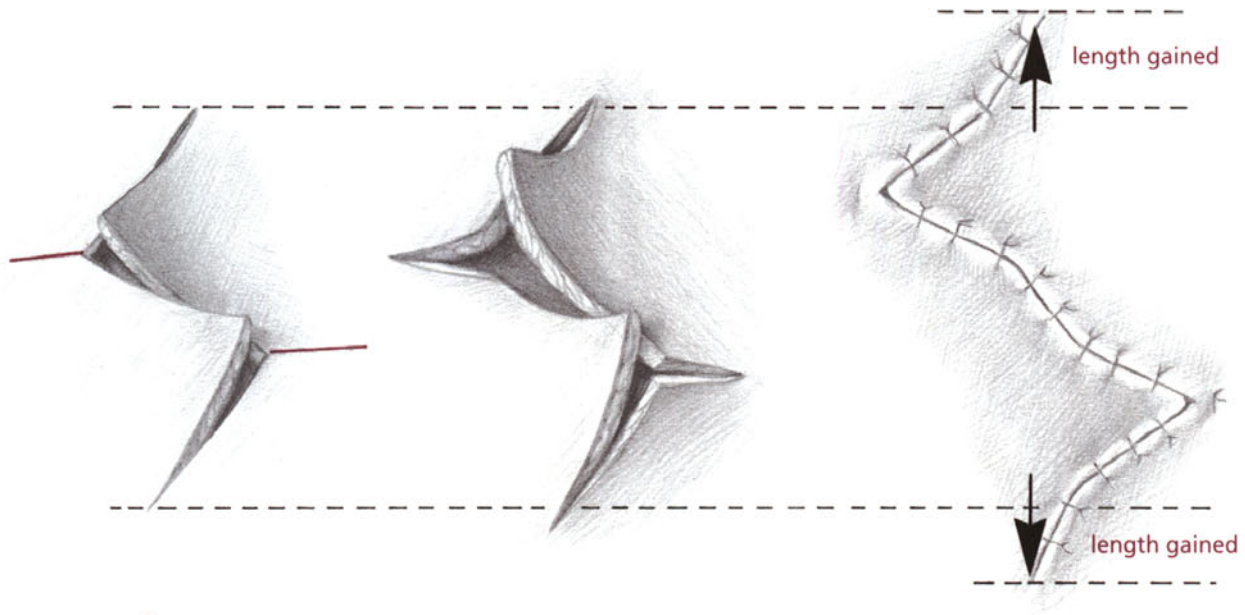


Fig. 49a–e. Y-V-plasties at the expanded Bruner incisions allow primary closure even if the fingers are completely extended (**a**). **b.** Contracture release by Y-V-plasty. **c.** The original Bruner incision with 90-degree angles at the apex. "Back cuts" on the contralateral side of the flap's tip. **d.** The length of these "back cuts" depends on the lengthening (gain in length) required to achieve unrestricted extension. Customarily, these "back cuts" are about $^1/_3$ of the lateral length of a Bruner flap. **e.** Lengthening can be achieved by advancing the flap's apex into the Y-shaped intersections. The tissue is advanced to provide more length. The Bruner flaps now have acute angles. Skin closure with intradermal Gillies corner-stitches to avoid necrosis at the apex. If undue tension complicates tip advancement, the transverse limbs of the V-Y-flap can be either skin-grafted or left open.



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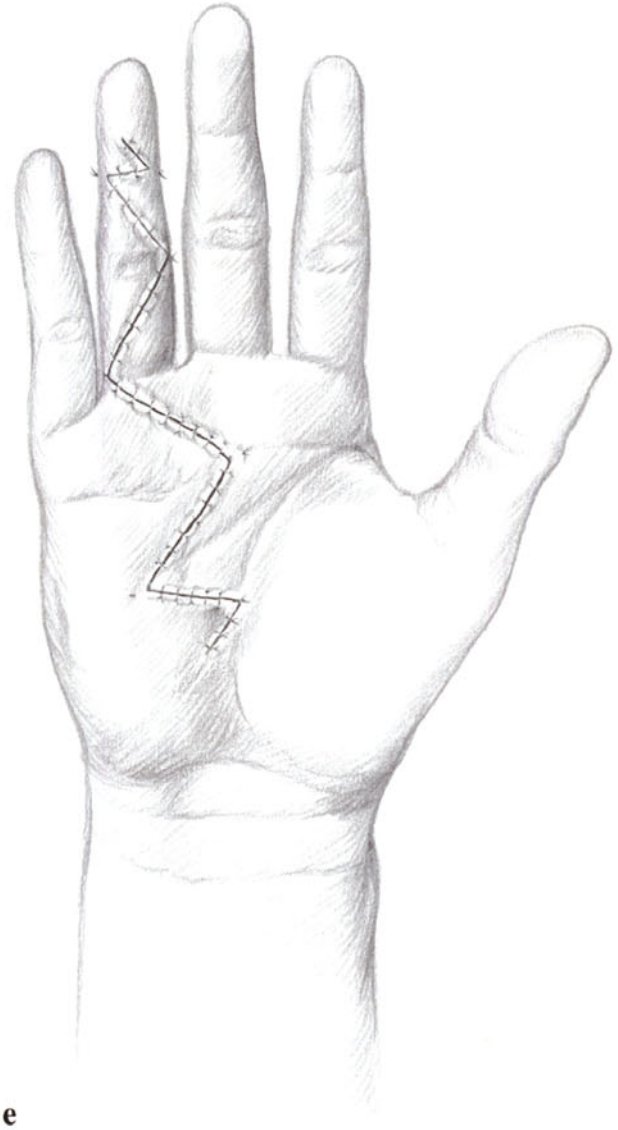

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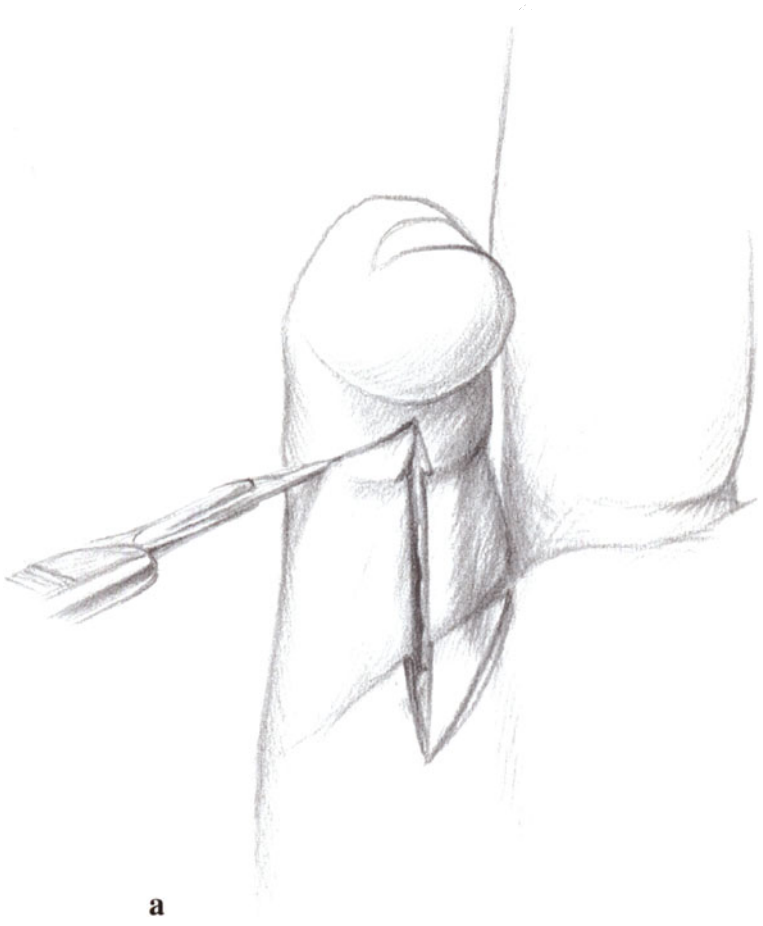
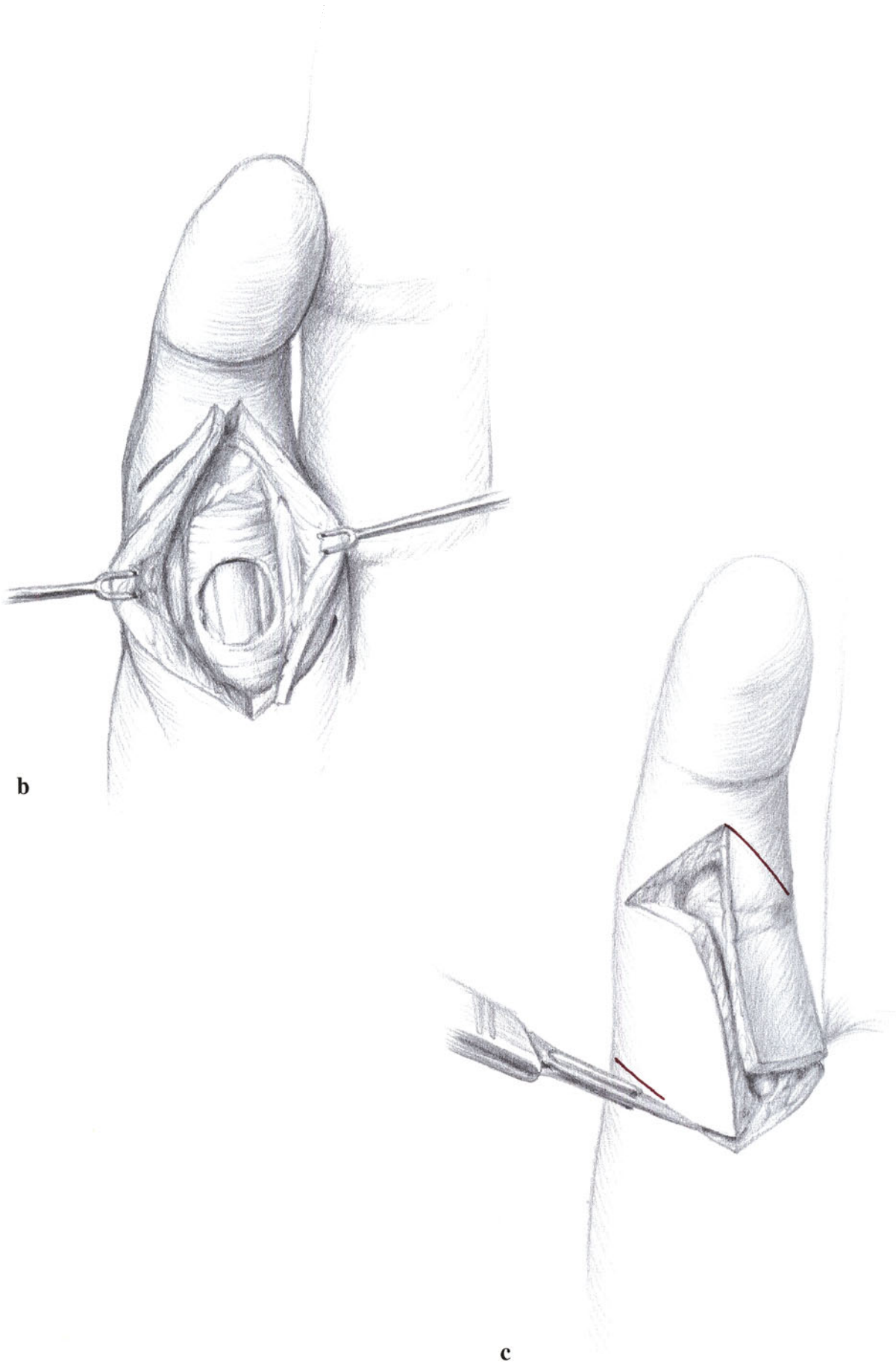


Fig. 50a–e. Z-shaped exposure according to Skoog in relapsing Dupuytren's contracture of the little finger. After excision and passive extension, the inelastic tendon sheath ruptured accidentally, exposing the flexor tendons at the level of the proximal phalanx. Two additional incisions were designed and cut next to the original Z-flaps, also releasing the severe scar contracture of the skin and leading to an unrestricted extension of the digit. The resulting bilateral transposition flaps were inserted as such. This helps to achieve tension-free soft tissue coverage over the opened flexor tendon sheath. Due to flap transposition, two well-vascularized defects result cranially and caudally. These are resurfaced by full thickness skin grafts.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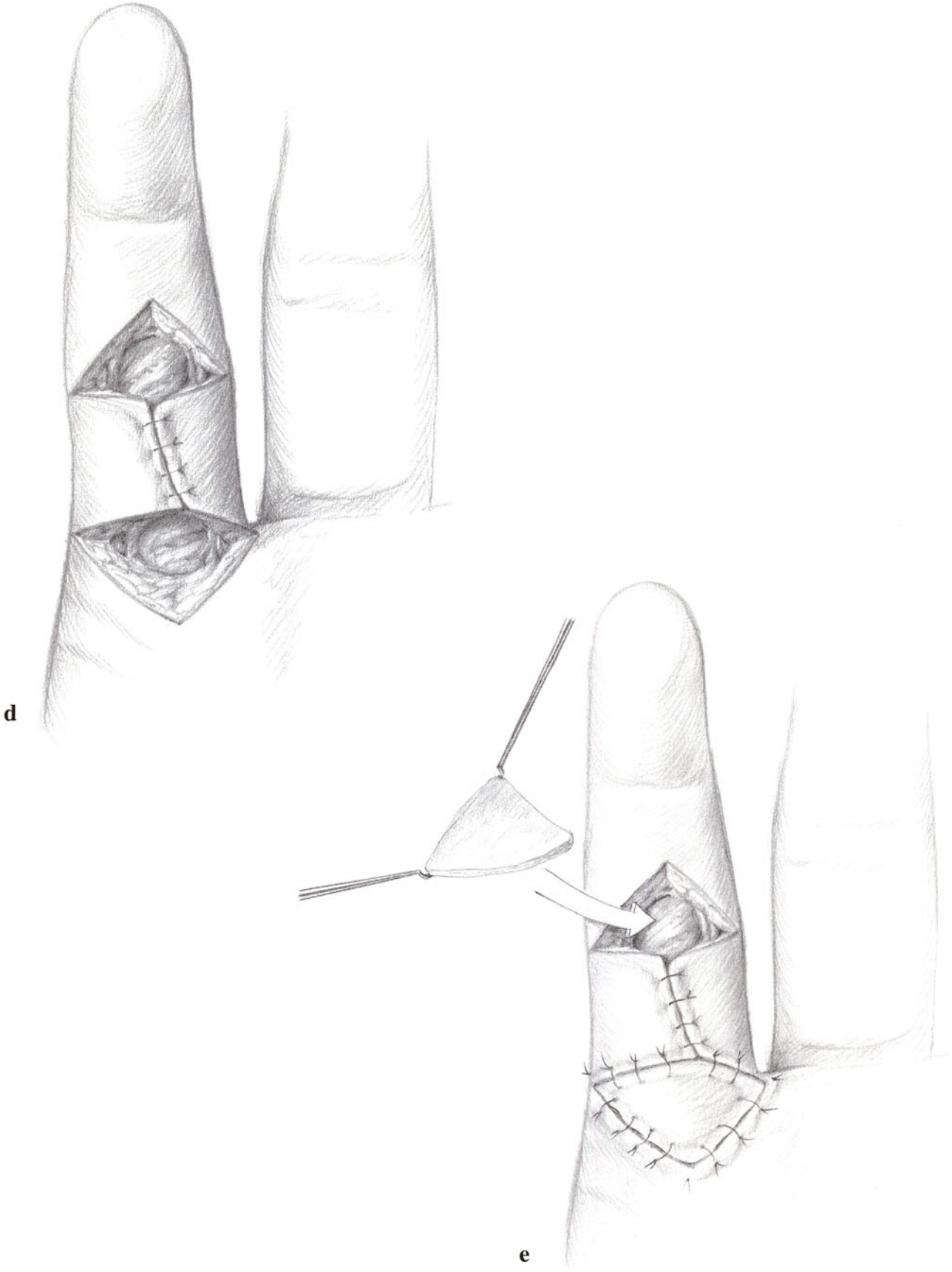
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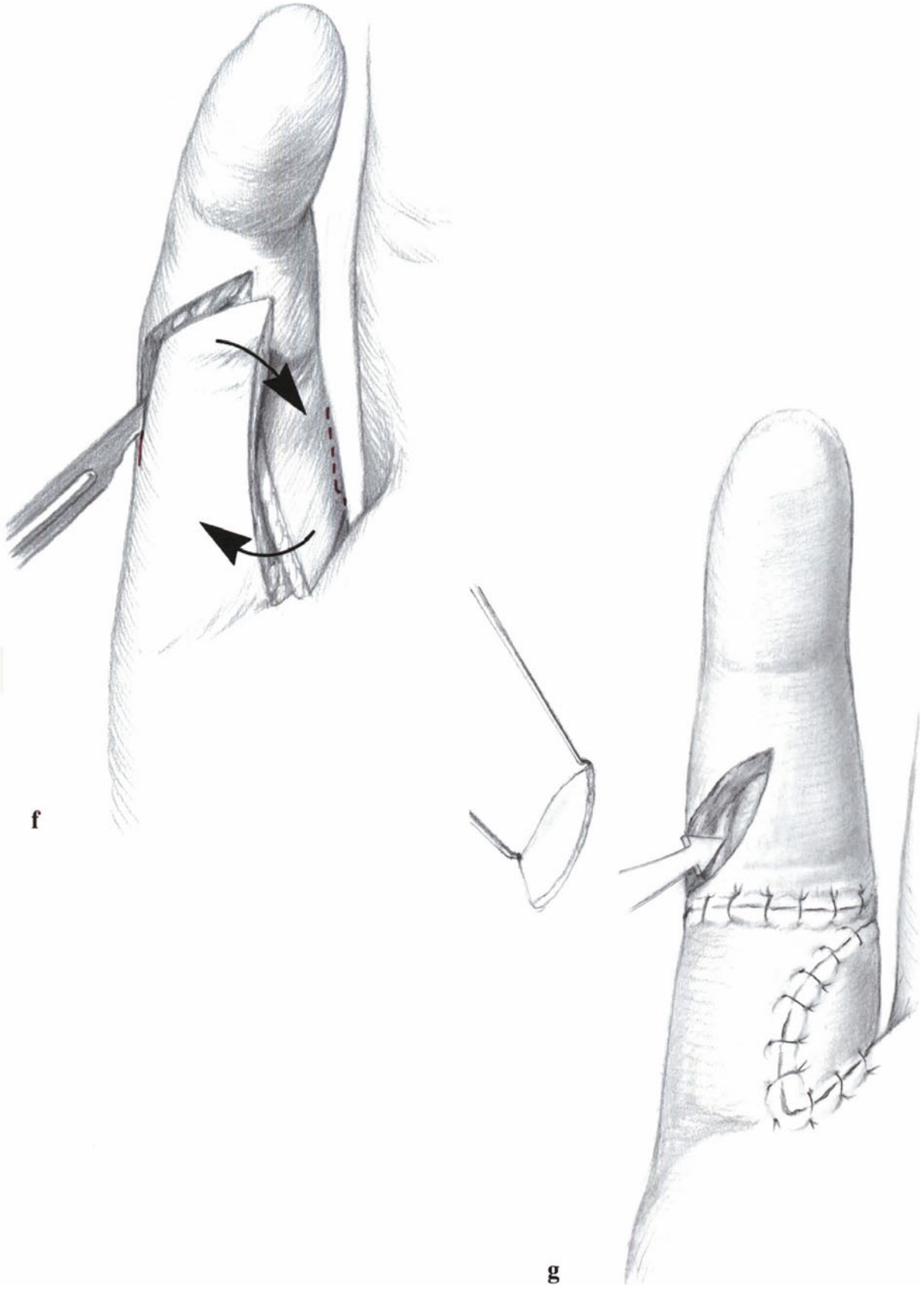

Fig. 50f, g. Modification of the above-mentioned procedure with primary use of large bilateral transposition flaps, which are cut to the mediolateral line of the digit. Tension-free approximation over the opening of the flexor tendon sheath. Resurfacing of the donor site by means of a full thickness skin graft.

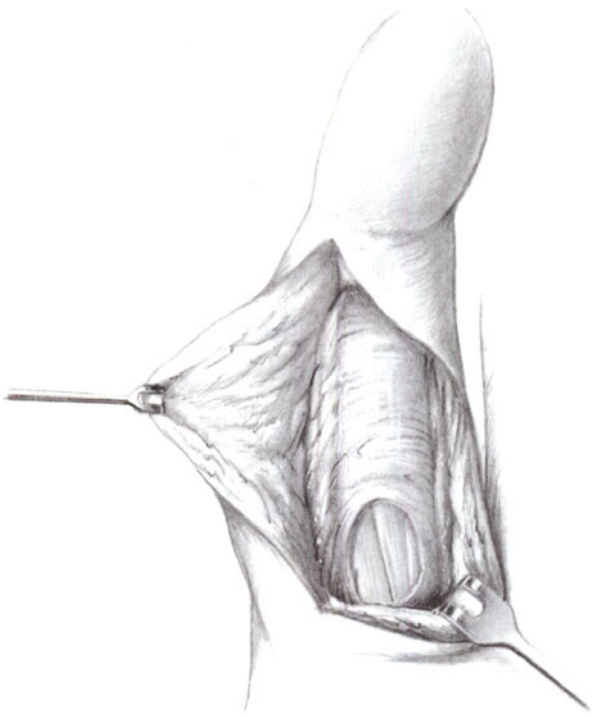

Fig. 51a. Laterodigital transposition flap used for the coverage of a soft tissue defect in the vicinity of the palmar digital crease.

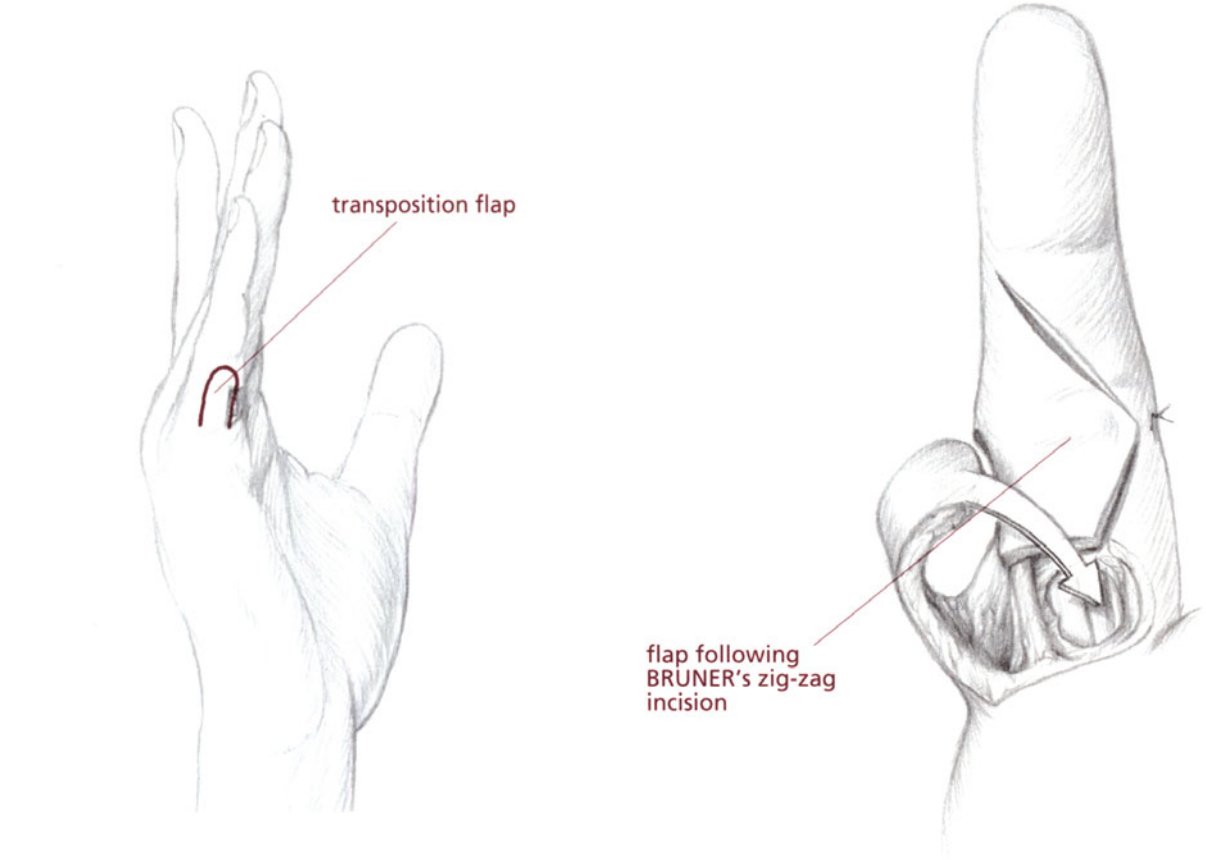

Fig. 51b. Using the soft tissue defect as a template, a tongue-shaped random transposition flap is planned over the mediolateral line of the finger. The skin flap is circumcised and raised from distal to central over the neurovascular bundle.

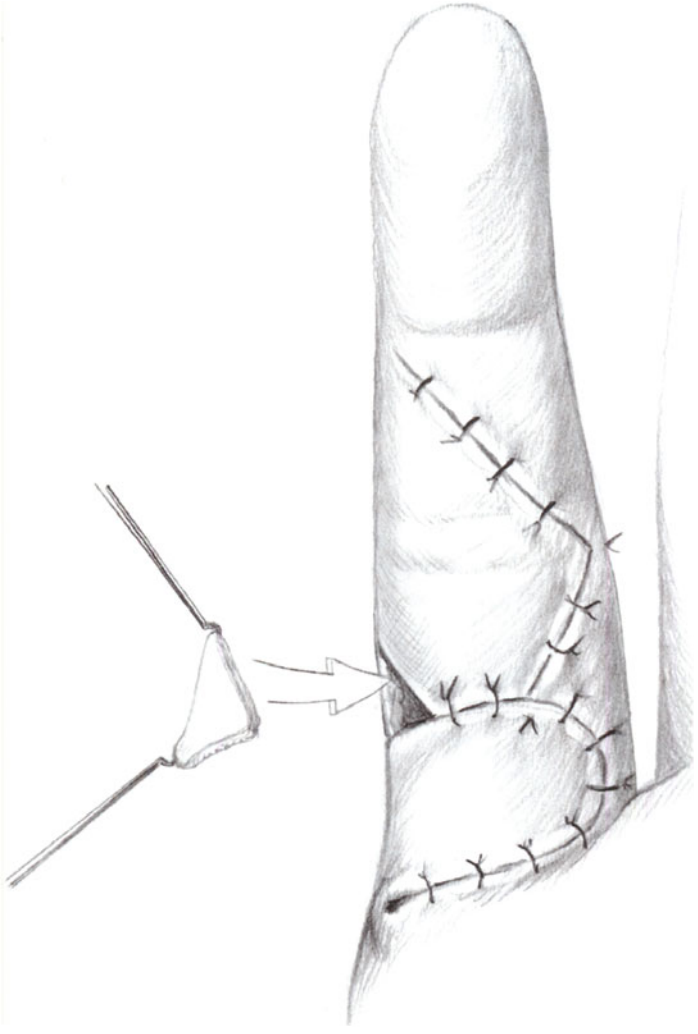

Fig. 51c. The tourniquet is released and the flap's viability checked. Careful hemostasis and tension-free transposition of the flap into the defect. The donor site is simultaneously covered with a full thickness skin graft.

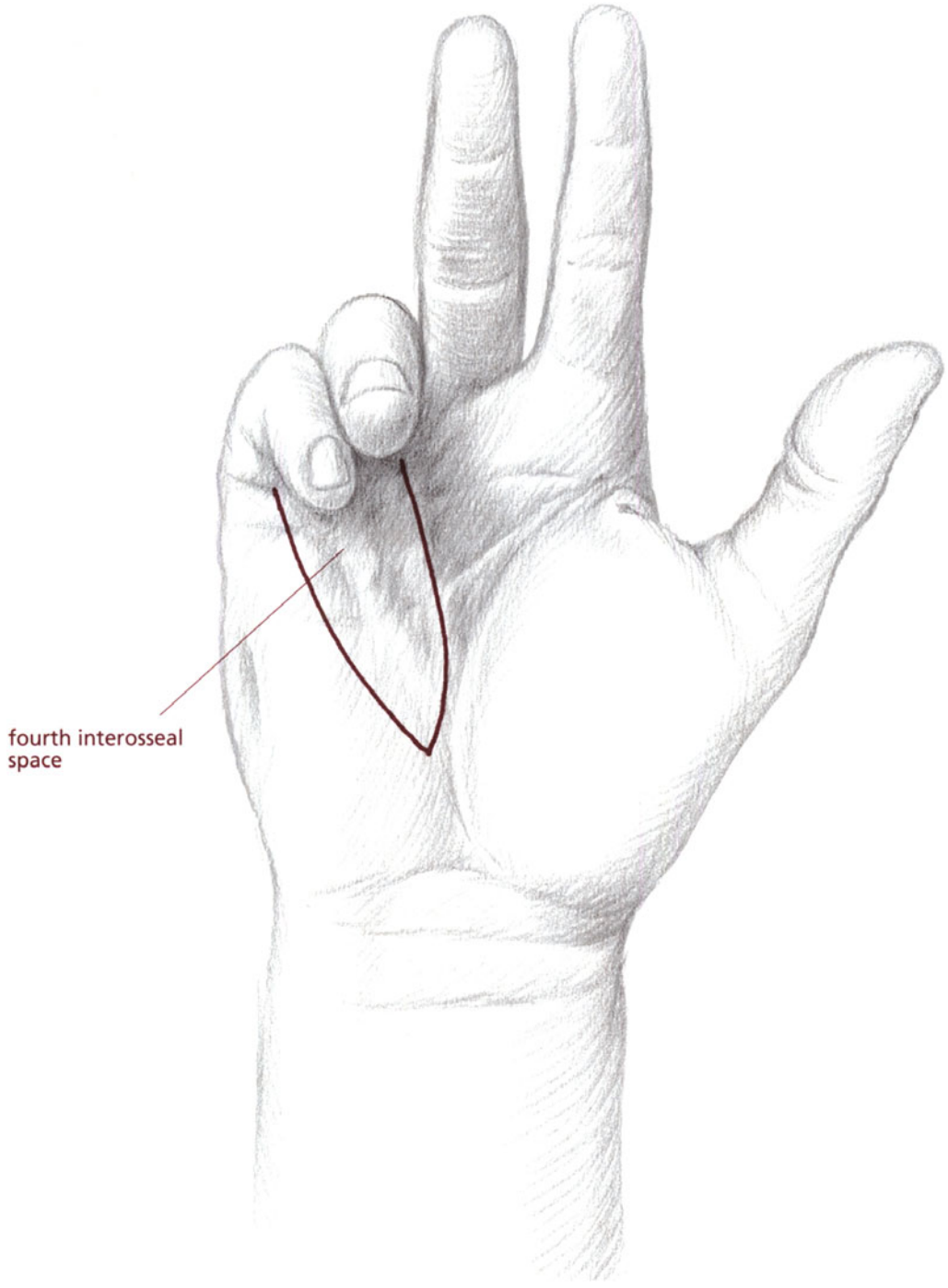

Fig. 52a. Retrogradely pedicled V-Y palmar flap according to Rider et al. (1998). V-shaped incision overlies both pretendinous cords of the contracted ring and little finger. The flap is designed in the fourth interosseal space around the direct perforating arteries of the monticuli.

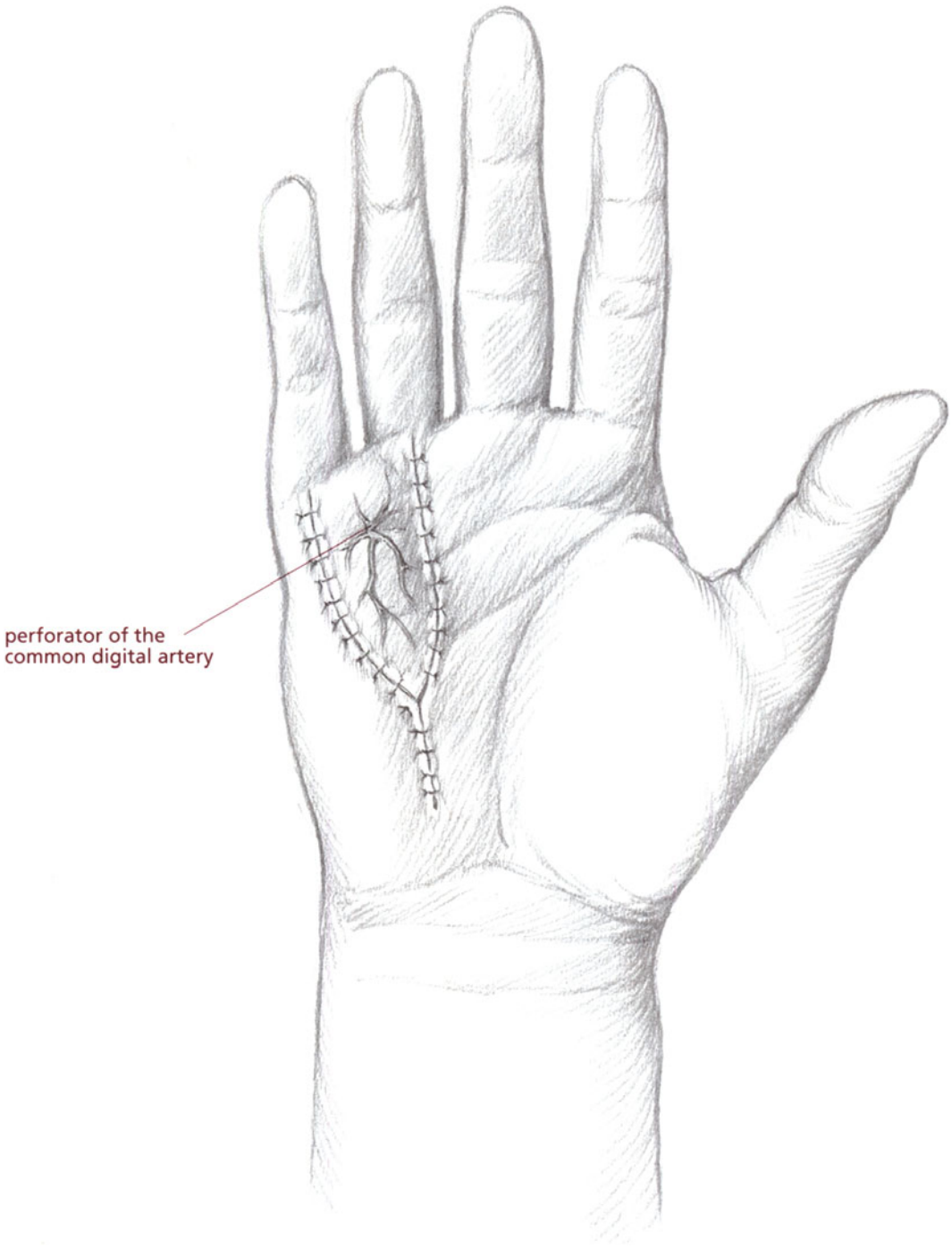

Fig. 52b. The Rider-flap is elevated distally. The desinsertion stops at the distal edge of the transverse fibers of the palmar aponeurosis to not impair the retrograde perfusion via perforators of the monticuli. After partial fasciectomy, the fingers can be extended without impairment. Because of the distalized flap, the Y-shaped primary closure in the palm succeeds.

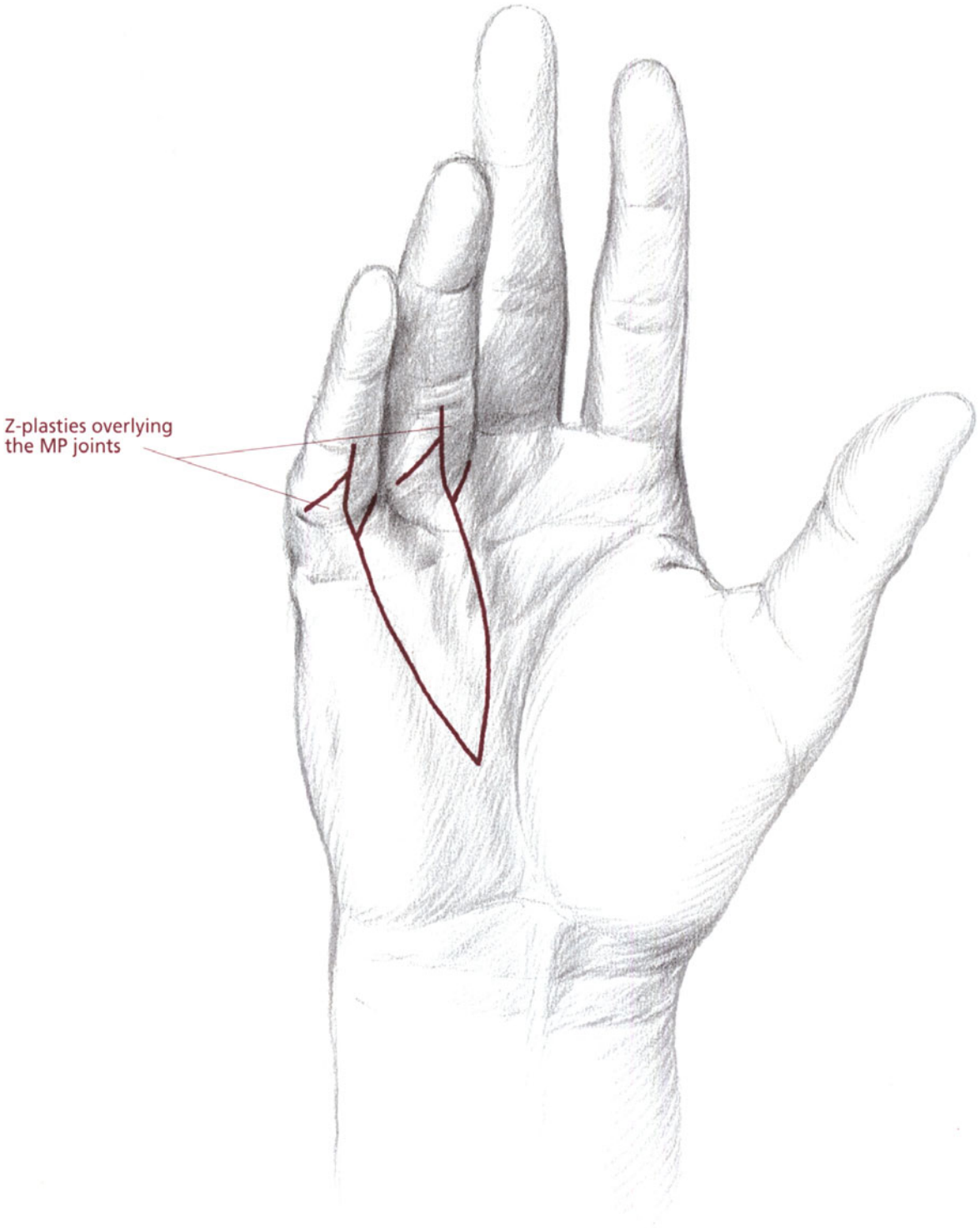

Fig. 52c. Expansion of the flap design toward the finger.

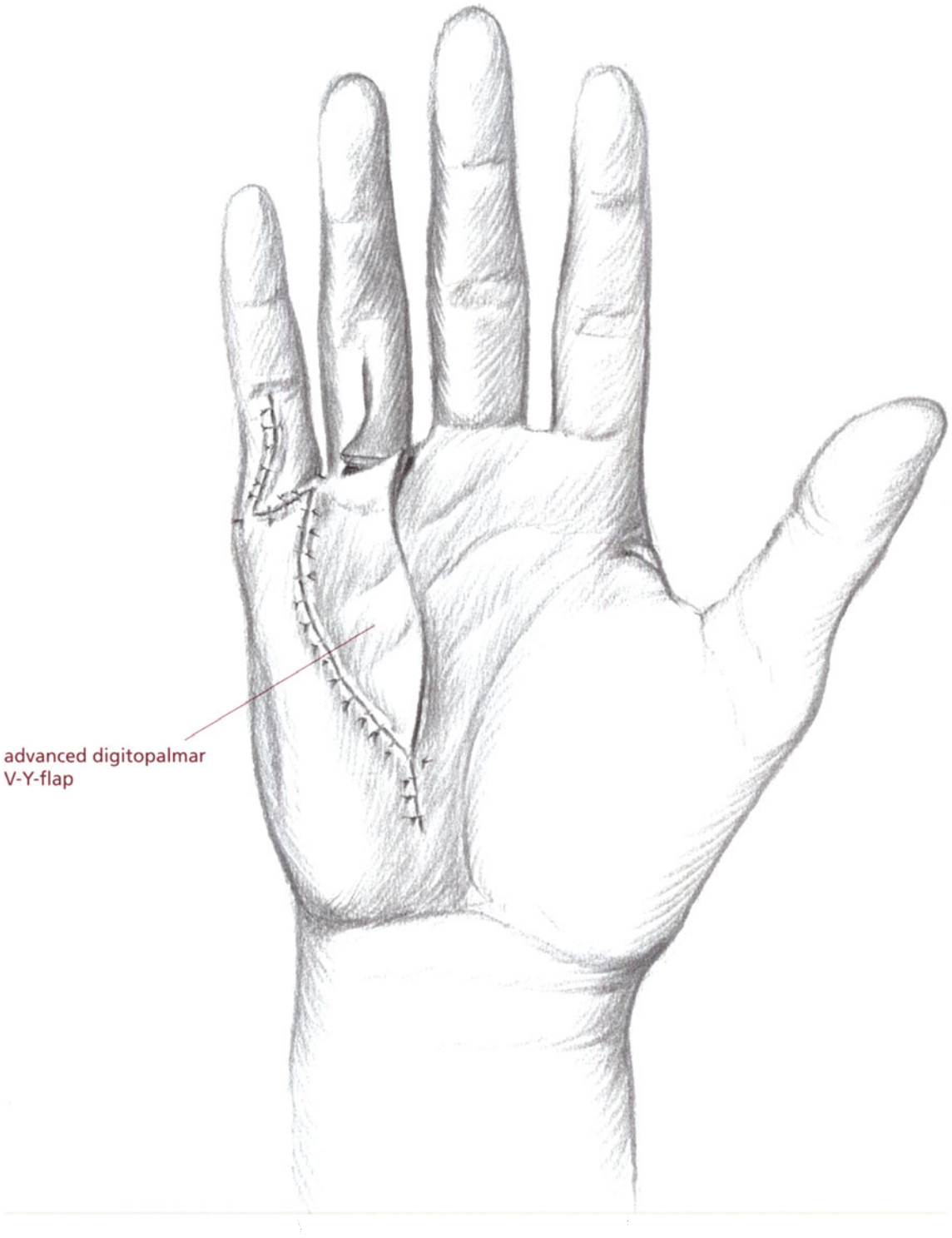

Fig. 52d. Insertion of the Rider-flap after distal advancement and Z-plasties above the metacarpophalangeal joints of the ring and little finger. Closure of the donor area by means of a V-Y-plasty. Full extension of the fingers is obtained.

For stable skin coverage of digito-palmar defects after fasciectomy and full extension of the finger, RIDER et al. (1998) recommends the V-Y palmar flap. It receives retrograde perfusion via perforators from around the monticuli. The V-shaped flap can be located either on the fingers or exclusively in the palm, or a combination of both. After advancement or scar lengthening, the donor area is closed in a Y-fashion. The different flap types are illustrated in Fig. 52a–d.

20.4.3 Heterodigital flaps

The previously mentioned laterodigital transposition flap, respectively the side finger skin flap, can also be used to cover defects on the neighboring finger, either as a random or as an axial pattern flap (RUSSELL et al., 1981).

If it is used as a heterodigital flap, it may have some disadvantages including iatrogenic syndactyly, when it is used very close to the commissures. Dysesthesias are another disadvantage, primarily when the flap is harvested as an axial pattern flap (WILHELM et al., 1997).

For even greater flap mobility, the dorsal incision may be extended more toward the interphalangeal web space.

The best-known heterodigital flap is the dorsal cross-finger flap, either as a common standard or a neurosensory flap.

This flap is indicated for deep soft tissue defects of the proximal, middle, and end phalanges of the index to little finger. The size of the flap is determined with a template that is transferred, for example, to the extensor side of the middle phalanx of the neighboring finger. With a broad flap base on the midlateral line, we dissect the cross-finger flap above the intact paratenon. The laterally based flap is transposed to the palmar side of the semiflexed finger, much like turning the page of a book, and sutured to the former defect without tension. A skin graft covers the donor site. At about two to three weeks the flap is detached (Fig. 53a–g).

The main disadvantage of the common cross-finger flap is the need for immobilization and a temporary loss of sensation. After a long time some protective sensibility may eventually be restored.

Reliable reneurotization can be achieved with a neurosensory cross-finger flap. The dorsal sensory branch opposite to the flap's base, which supplies the skin on the extensor side of the middle phalanx, is incorporated into this flap. To achieve an effortless coaptation with the contralateral digital nerve stump on the palmar side, the dorsal sensory branch has to be dissected over an additional length ("tail") of 15–20 mm (BERGER et al., 1975). The procedure resembles the standard cross-finger variant but includes an additional microsurgical suture (coaptation). As the nerve suture is located opposite of the flap's base, it cannot be unintentionally divided when the flap is detached.

The cross-finger flap is contraindicated in Raynaud's disease, joint stiffness, and reflex sympathetic dystrophy (WILHELM et al., 1997).

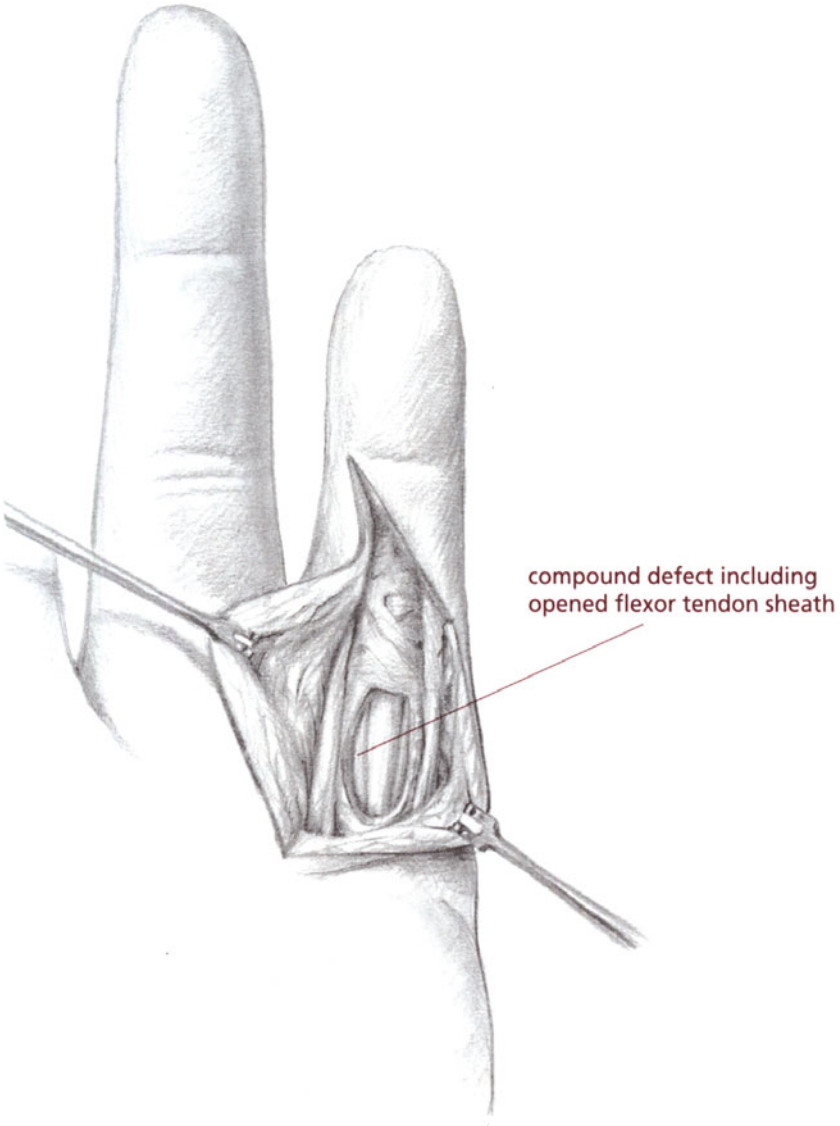

Fig. 53a. Soft tissue defect with opened flexor tendon sheath over the palmar proximal phalanx of the little finger following partial fasciectomy.

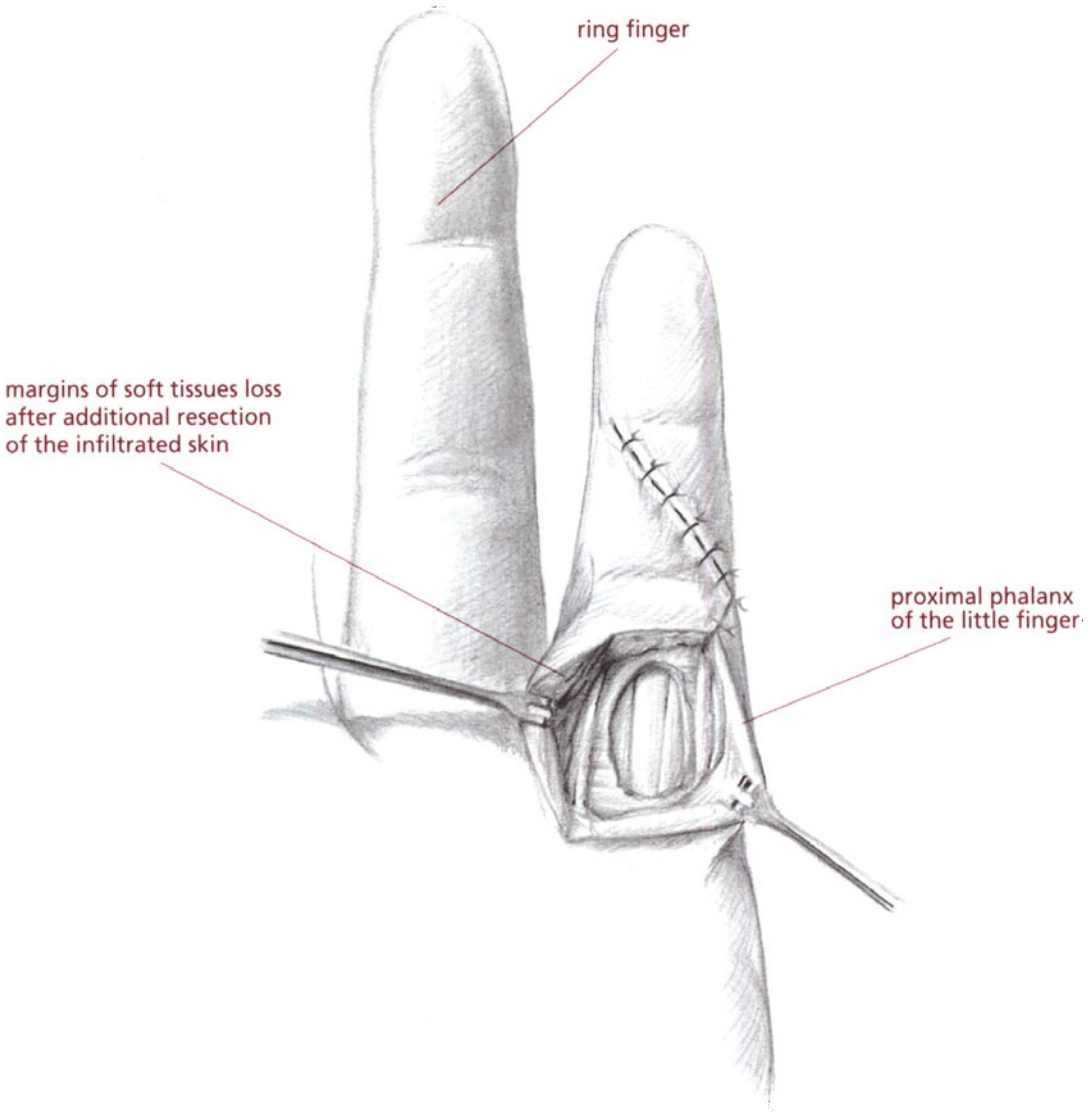

Fig. 53b. Flap design according to template.

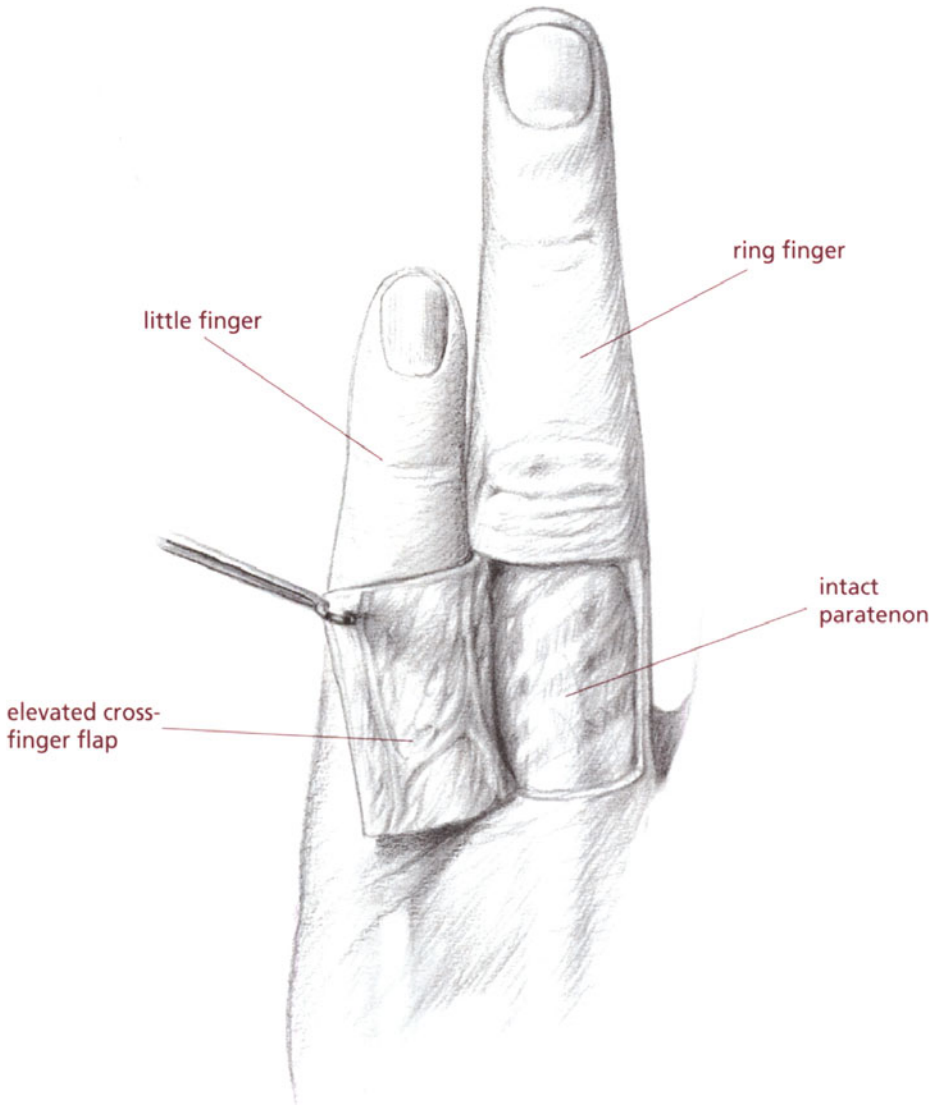

Fig. 53c. Harvest site is the dorsal aspect of the proximal phalanx of the ring finger. The maximal length-to-breadth ratio is 3:1. U-shaped circumcision of the planned flap with a broad base on the ulnar side for secure perfusion. Level of dissection is the paratenon covering the extensor tendon, where a well-defined tissue plane will be found.

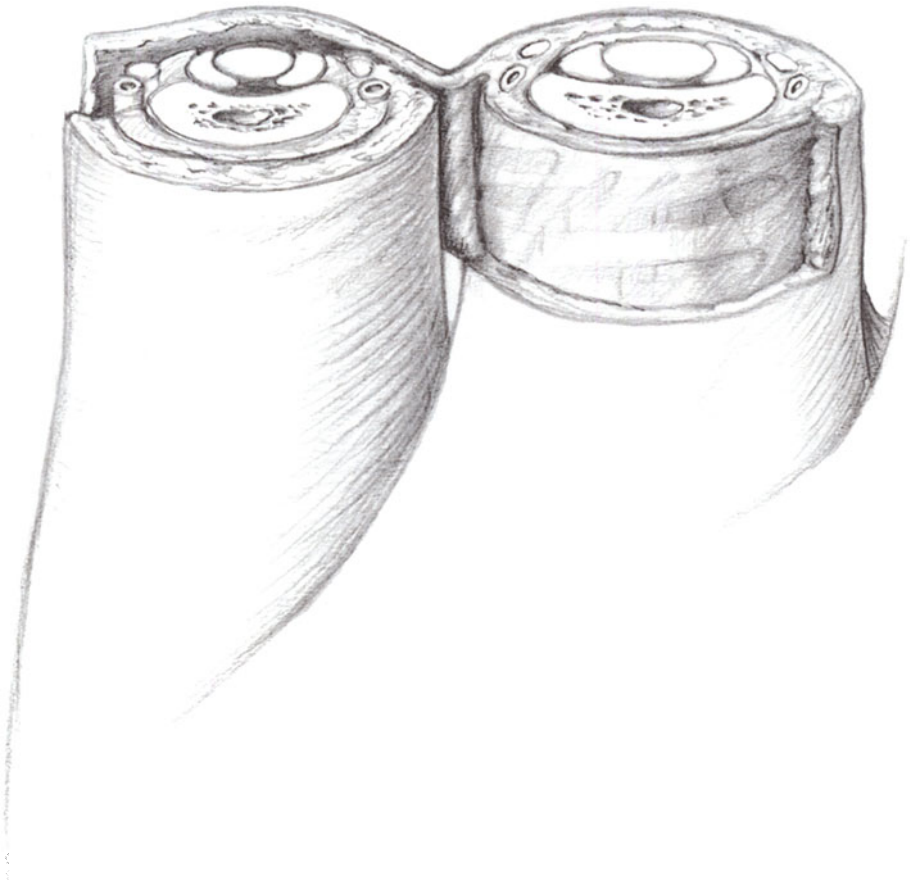

Fig. 53d. Principle of the cross-finger flap in cross section of two fingers. From the dorsum of the adjacent finger (depicted on the right), a mediolaterally-based, random cross-finger flap is reflected to the palmar aspect of the neighboring finger, much like turning the page of a book, to cover a palmar soft tissue defect. The harvest site is covered by a split thickness skin graft.

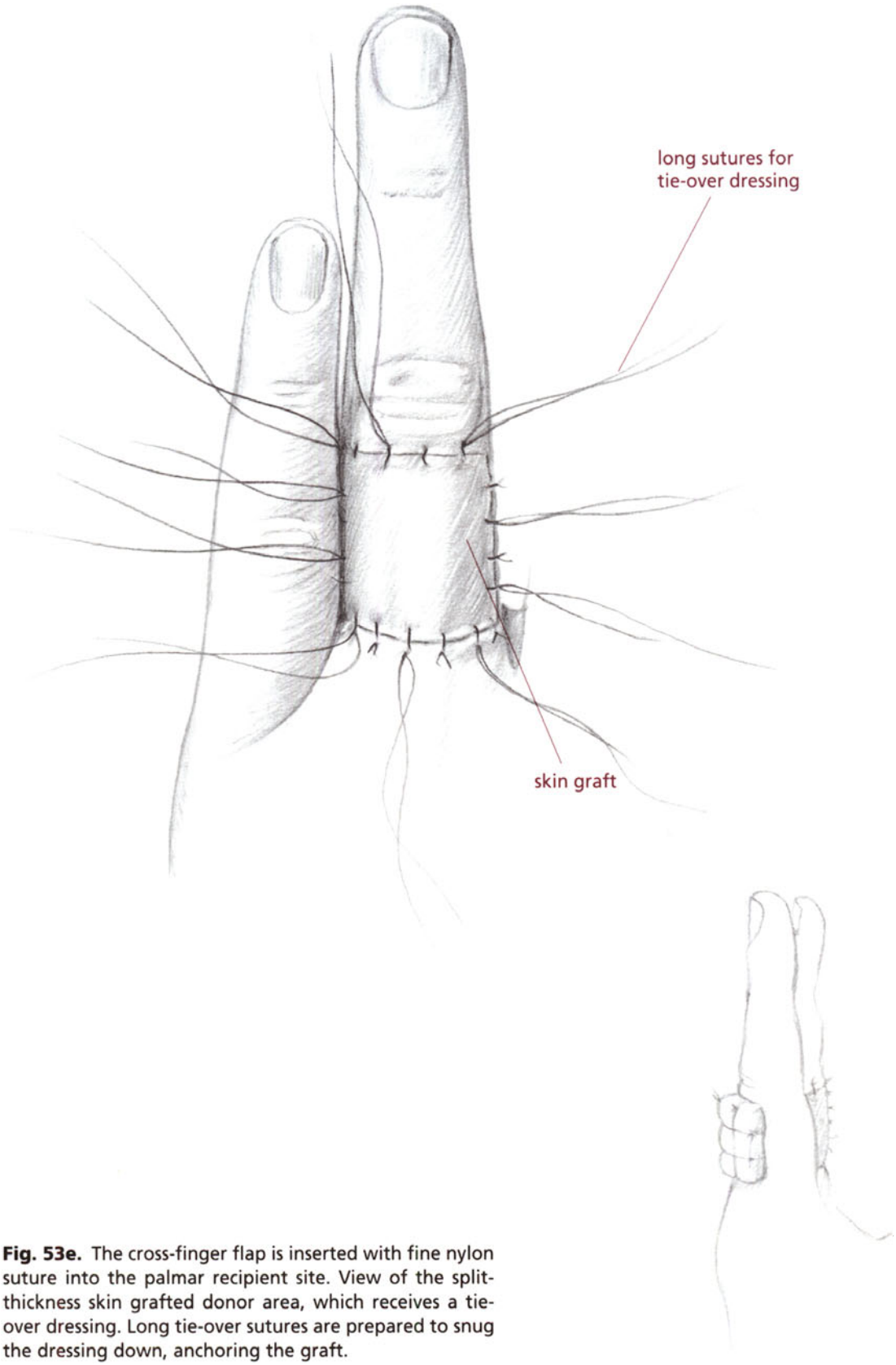

Fig. 53e. The cross-finger flap is inserted with fine nylon suture into the palmar recipient site. View of the split-thickness skin grafted donor area, which receives a tie-over dressing. Long tie-over sutures are prepared to snug the dressing down, anchoring the graft.

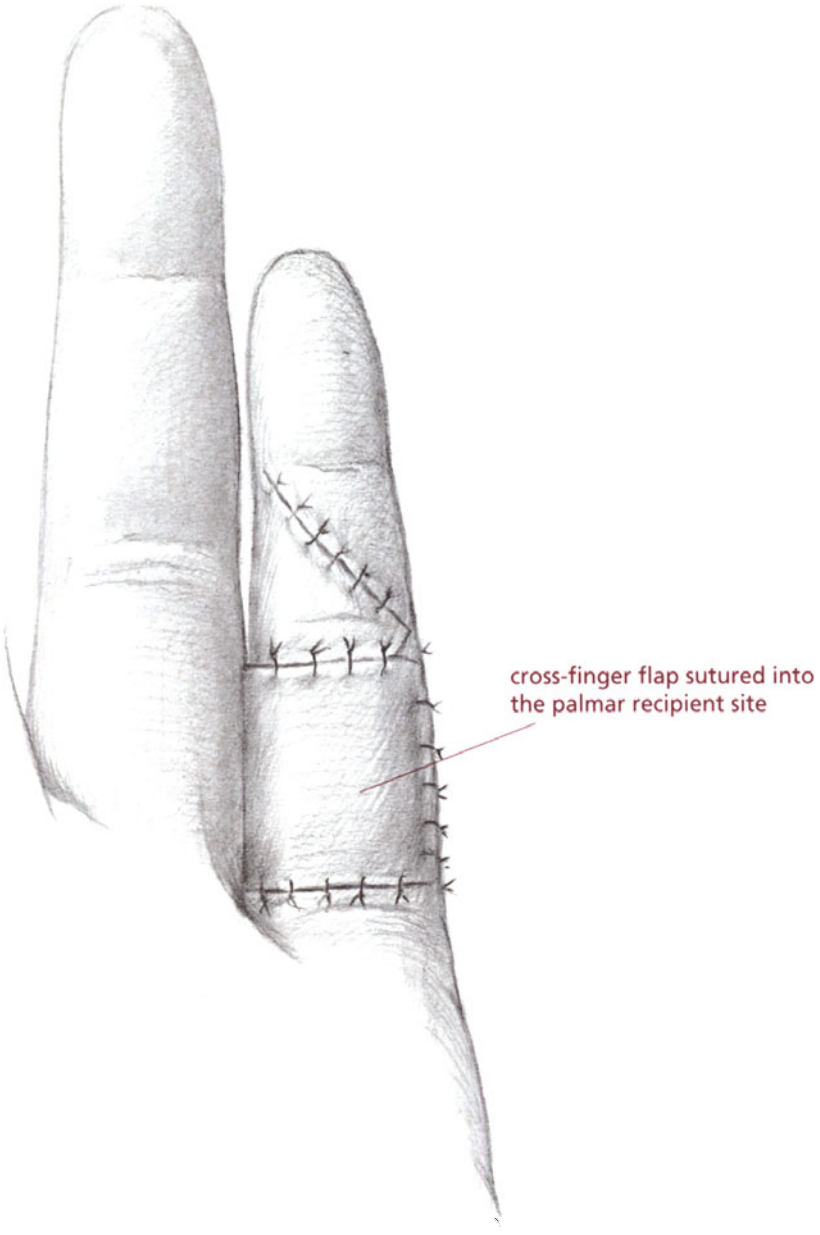

Fig. 53f. View on the palmar aspect. The cross-finger flap resurfaces the soft tissue defect tension-free.

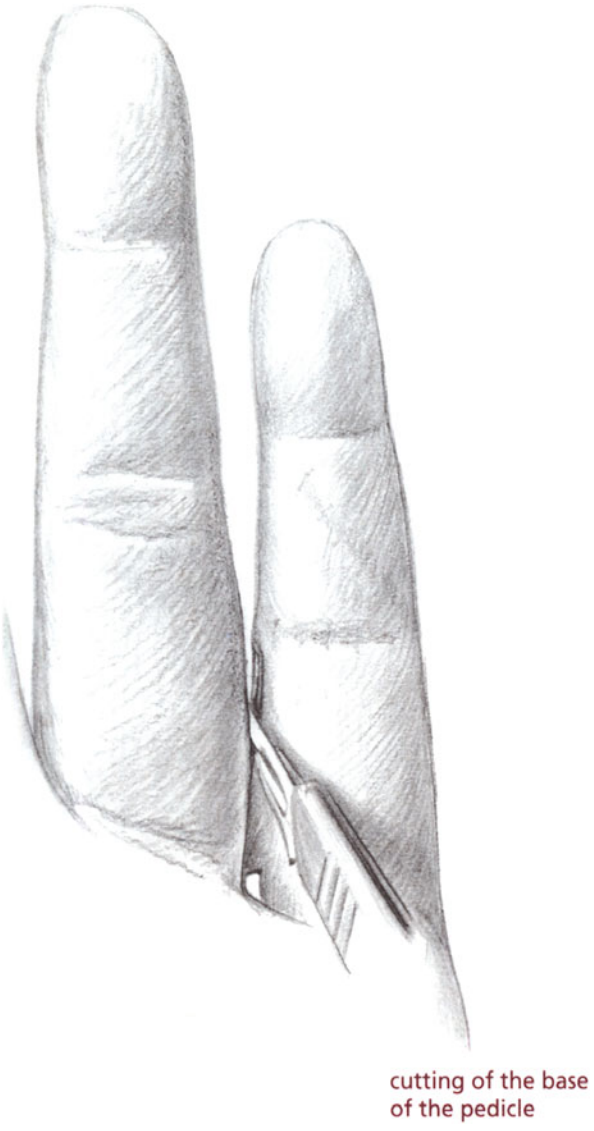

Fig. 53g. After two to three weeks the base of the pedicle is divided and both fingers are detached from each another (the pedicle is divided under local/regional anesthesia).

20.4.4 Free tissue transfer

Free "flow-through" flaps as vascular conduits can save fingers affected by relapses of Dupuytren's disease if they develop acute ischemia.

Their advantage is simultaneous revascularization and soft tissue coverage.

Another advantage is that no additional vascular interposition grafts need to be harvested. The match of the arterial calibers seems better than that of veins, and the number of microvascular anastomoses is reduced. This is particularly true if one considers that a supplementary free tissue transfer would be needed, in addition to the conventional venous graft used to bridge the missing vascular segment.

Owing to the perfect match of the vascular caliber, arterialized venous flaps and temporo-parietal fascial flaps covered with full thickness grafts (Fig. 54a–d) are particularly suited to this, when filet flaps harvested from the non-replantable amputated digits are not available. The dorsalis pedis flap can be recommended only with some reservation (BRANDT et al., 1996).

The options for soft tissue closure presented here are only a small part of the armamentarium of locoregional and free flaps. Some of the choices simply reflect the author's personal preferences. This compendium does not intend to list all the options available and cannot replace encyclopedic literature on flap surgery.

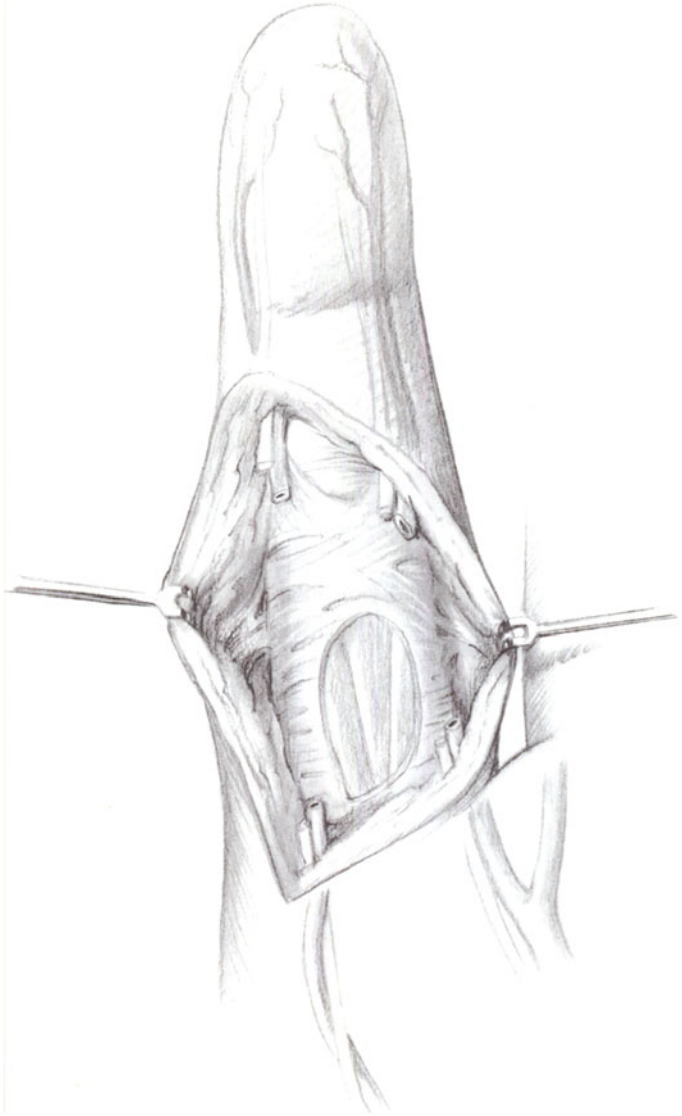

Fig. 54a. Palmar soft tissue defect with segmental loss of the digital arteries over an extended distance; viability of finger endangered.

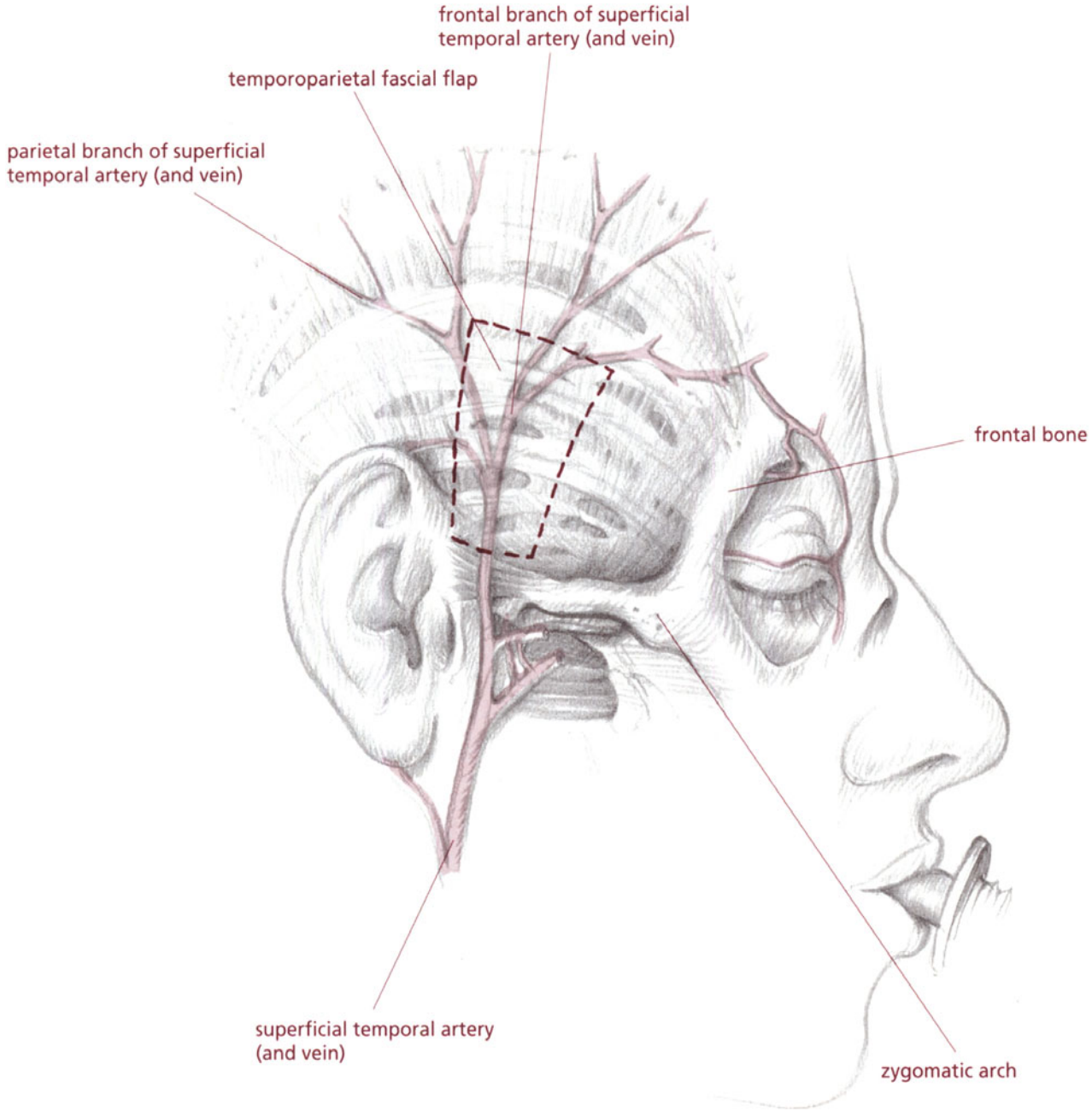

Fig. 54b. Raising of the free temporo-parietal fascial flap with axial vascular pedicle (superficial temporal artery and vein). The superficial temporal fascia is elevated off the deep temporal fascia, which covers the temporalis muscle. The frontal vascular branch is most often used for the planned conduit flap.

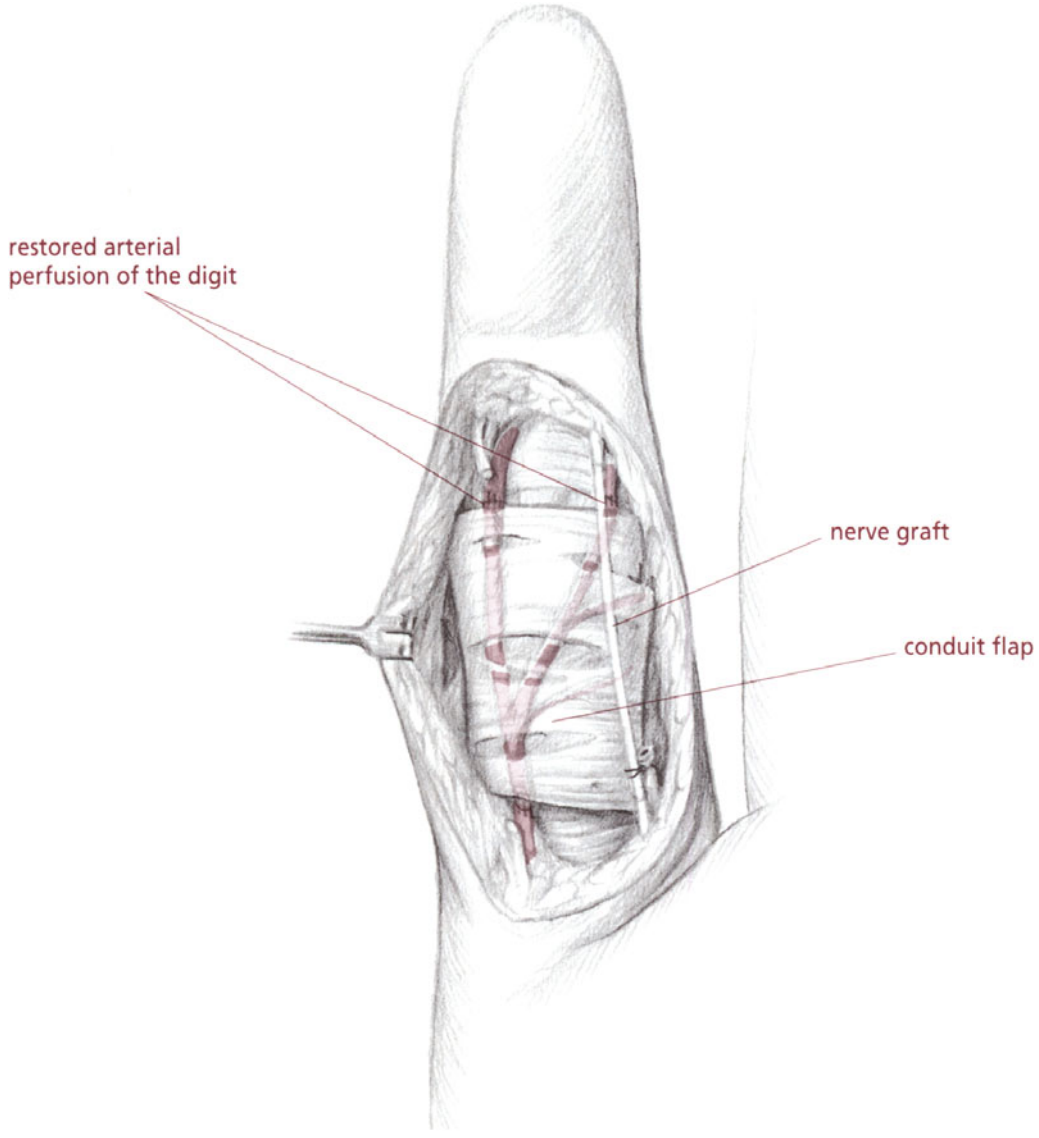

Fig. 54c. Because of the good arterial caliber match, the temporo-parietal fascial flap represents an ideal "flow through" flap to revascularize fingers. The segment of the superficial temporal artery can be used to bridge the vascular defect of the digital artery. It simultaneously supplies the fascial flap used for coverage. Venous return is via subcutaneous veins of the dorsum. A full thickness skin graft resurfaces the fascial flap.

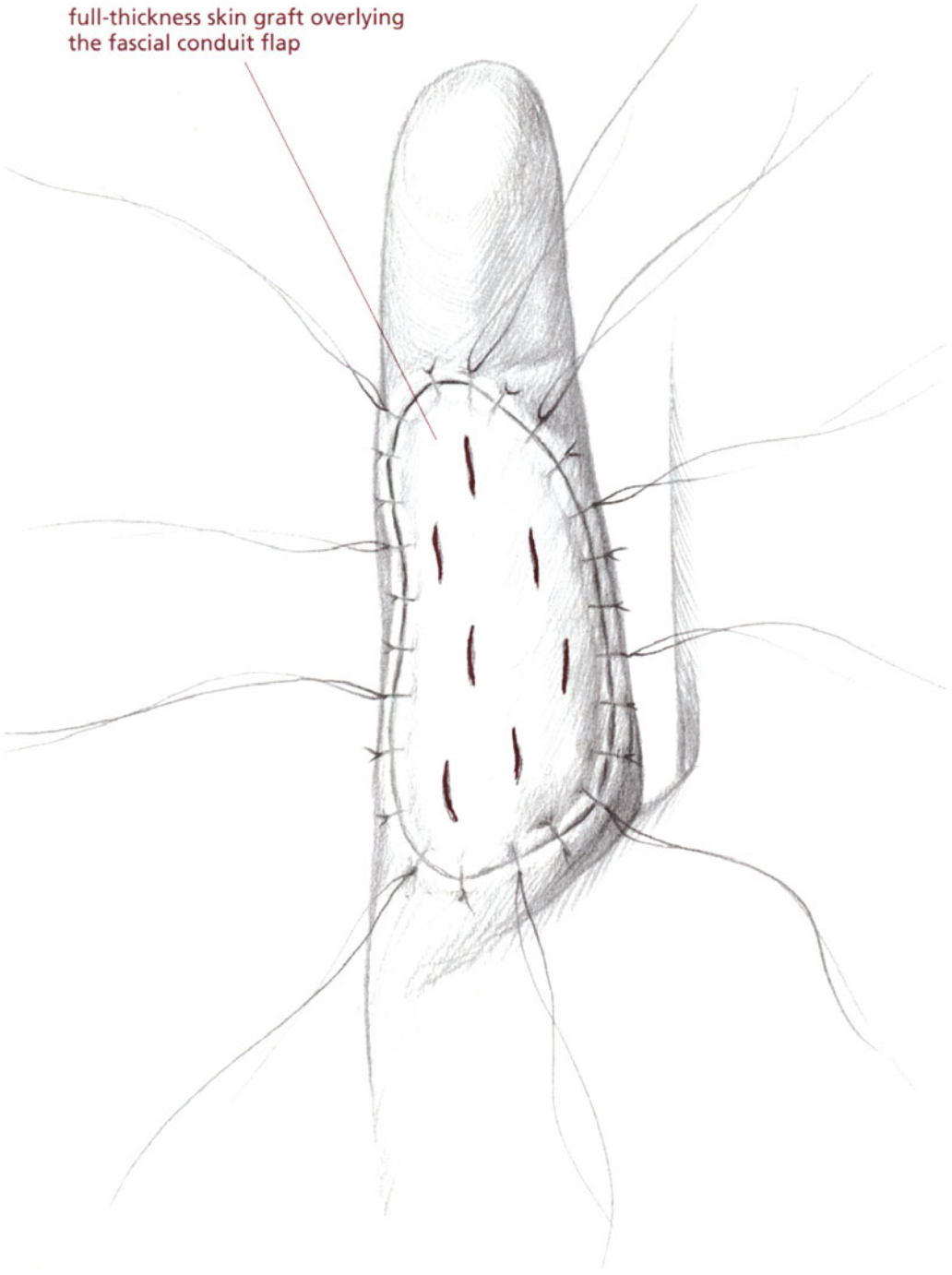

Fig. 54d. Scarified full-thickness skin grafts cover the fascial flow through flap.

21 Additional measures to improve hand function

21.1 Fixed flexion deformity at the interphalangeal joints

Even after prolonged episodes of flexion, full function of the metacarpophalangeal joint can usually be restored. That does not hold true for the proximal interphalangeal joint. Residual contractures of the proximal interphalangeal joints up to 30 degrees do not impair everyday tasks (CROWLEY et al., 1999).

Arthrolyses, corrective osteotomies, digital arthroplasty of the proximal interphalangeal joints, arthrodeses, various extension methods, and amputation or exarticulation are palliative measures to improve hand function if a fixed flexion deformity of the PIP joint persists after fasciectomy (ANDREW, 1991).

21.2 Extension methods

HODGKINSON's Pipster extension device (1994) is based on the MESSINA technique. In comparison to its predecessor, the device is miniaturized and an advancement. The Pipster procedure aims to straighten PIP joints, which are affected by fixed flexion contractures, over 10 days to four weeks. This primary step should be followed by a fasciectomy. HODGKINSON (1994) places two percutaneous Kirschner wires, one transversely across the base of the proximal phalanx and the second one into the head of the middle phalanx to fixate the bone. Bilaterally, a modified miniaturized external fixateur device is attached with one screw each. A small silicone spacer is interposed to avoid skin injury. A nut is turned to gradually separate the two fixation points over at least 10 days. Thus a severe flexion contracture can be reduced to a moderate extension deficit of about 45 degrees at the proximal interphalangeal joint. This facilitates the planned fasciectomy. In a pilot study this procedure apparently helped to avoid the threat of amputation of three fingers among seven fingers operated upon (n = 5 patients).

With regard to its design and use, the Pipster device resembles the Verona apparatus (CITRON et al., 1998).

We consider the pneumatic extension procedure described by PIZA-KATZER et al. (2000) primarily a preoperative adjunct, and not a useful technique for treating residual interphalangeal contractures of PIP joints.

21.3 Remarks on teno- and arthrolysis

The sustained flexion contracture in Dupuytren's disease results in secondary changes to the extensor apparatus. These contractures may affect all three digital joints and can be successfully treated by tenotomies at varying levels (HUESTON, 1985). In case of hyperextension of the distal interphalangeal joint despite adequate fasciectomy, the author recommends tenotomy of Landmeer's oblique retinacular ligament. To correct the buttonière-like flexion contracture of the proximal interphalangeal joint induced by fibromatosis, Fowler's tenotomy is recommended, and the central slip of the interosseus extensor tendon serves as a "living splint." To resolve residual contractures, especially of the proximal interphalangeal joint of the little finger, tenotomies of the lumbrical muscles and the abductor digiti muscle are important.

In Dupuytren's disease, extension deficits due to fibrosis and secondary changes of the intrinsic muscles, as well as pronounced contractures of the proximal interphalangeal joints are a frustrating problem for hand surgeons (Fig. 55a–d).

WEINZWEIG et al. (1996) retrospectively compared the functional results after severe flexion contractures of the proximal interphalangeal joint

(> 60 degrees), with and without capsuloligamentous release. In spite of capsulotomy and desinsertion of the check reins, no improvement was shown statistically compared with a noncapsulotomy group. The complication rate after capsulotomy was 27% compared with 19% in cases where the capsule remained untouched.

As it is known that arthrolysis with temporary Kirschner wire fixation does not yield better long-term results compared with conventional capsulotomy, McGrouther (1999) recommends a generous interpretation of the indication for arthrodesis.

Breed et al. (1996) claim, however, that exclusive, passive manipulation yields better functional results in residual contractures of the proximal interphalangeal joints and a lower complication rate compared with more aggressive surgical intervention.

Summarily, the literature agrees that after all treatment options have been exhausted, a residual contracture of up to 45 degrees is tolerable.

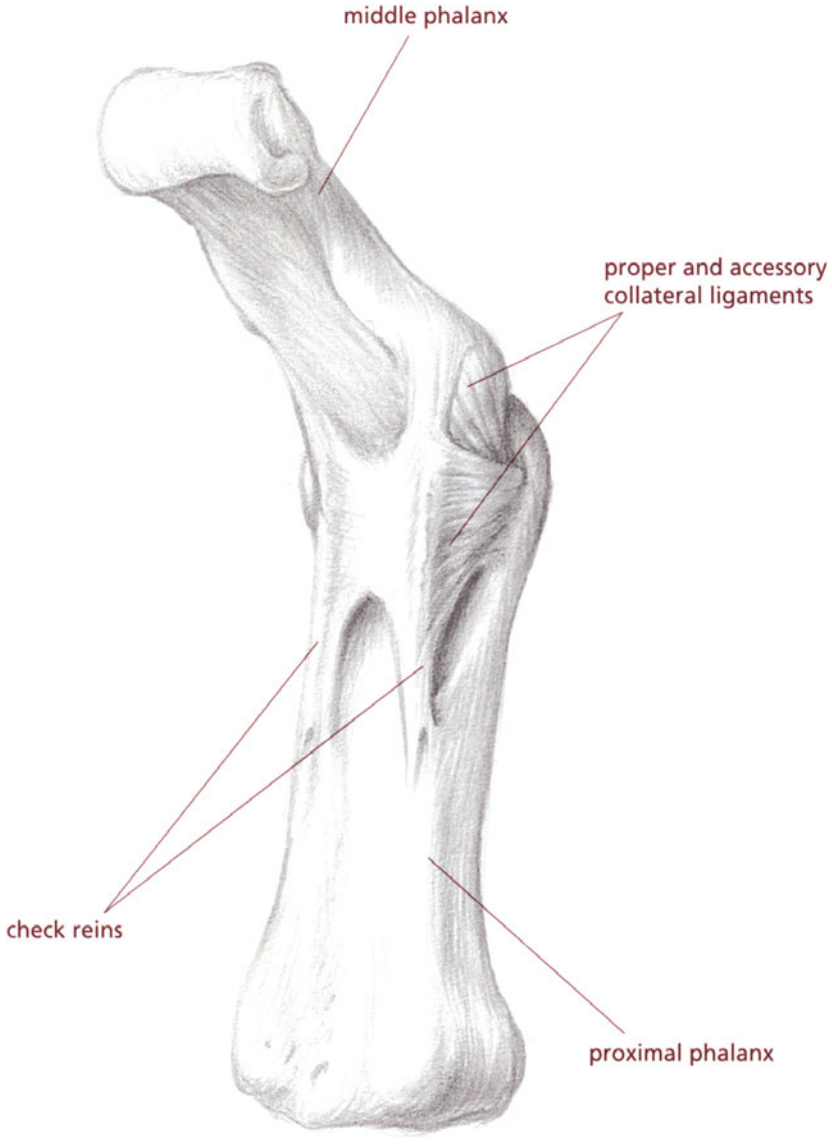

Fig. 55a. Scheme of the persistent secondary PIP joint contracture.

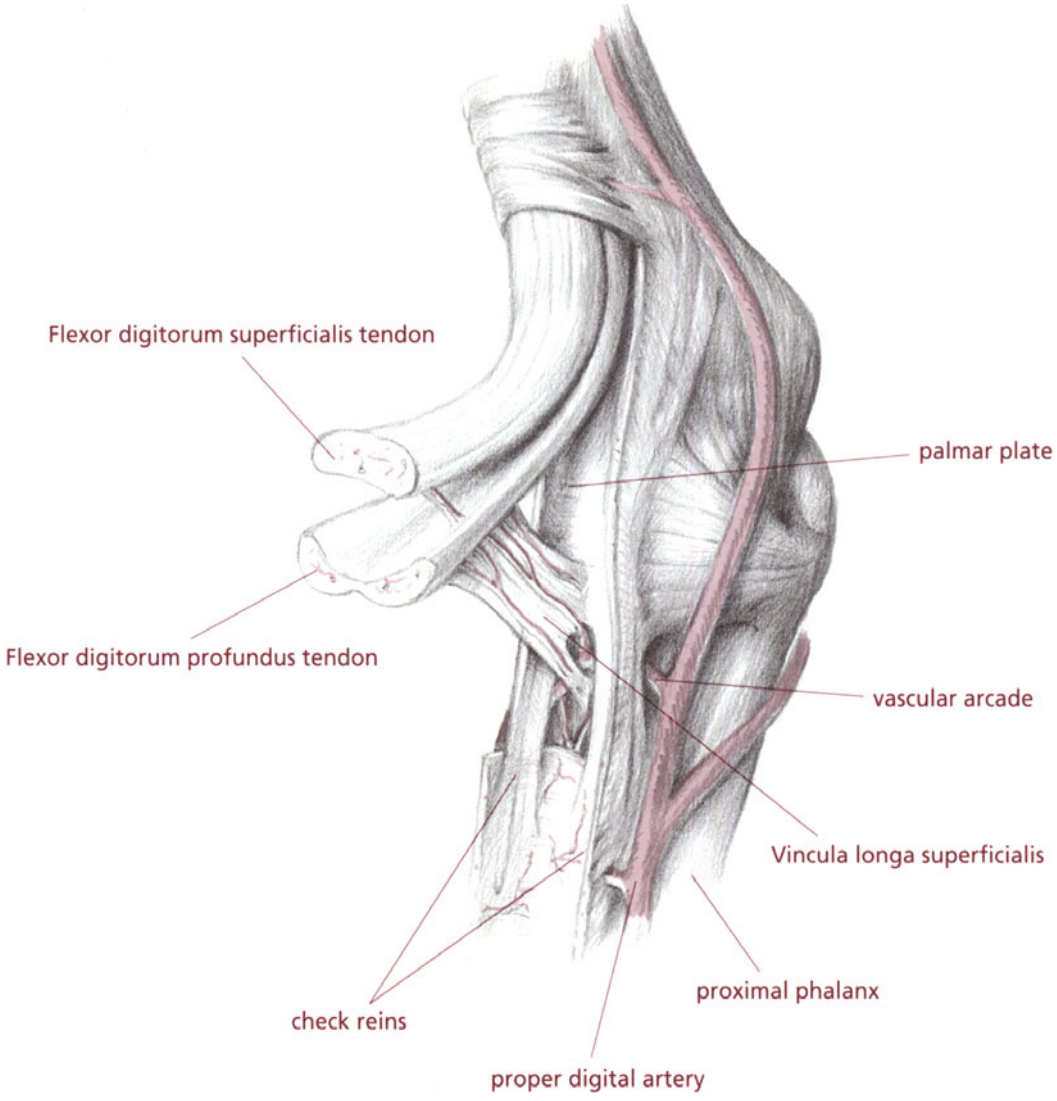

Fig. 55b. The check reins are swallow tail-like connective tissue "ligaments", which proceed between the palmar plate and their plane of insertion at the ulnar and radial assembly lines (bony ridges) of the shaft of the proximal phalanx. After exposing the bilateral proper digital arteries and the transverse communicating branch (vascular arcade) of the vincular system below the check reins, these bands are detached from the palmar plate's base or even completely resected.

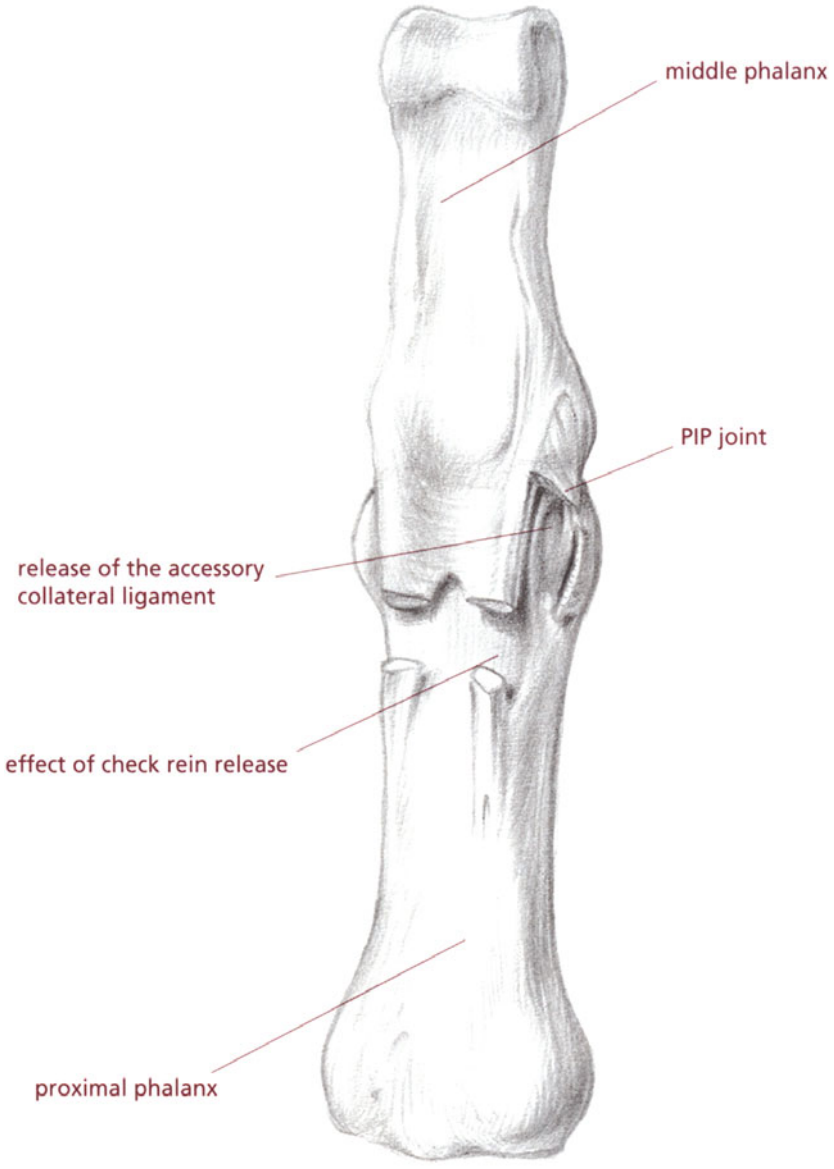

Fig. 55c. Persistent secondary contractures of the PIP joints are treated by first resecting the check reins. In case of residual retraction, the accessory collateral ligaments are also divided.

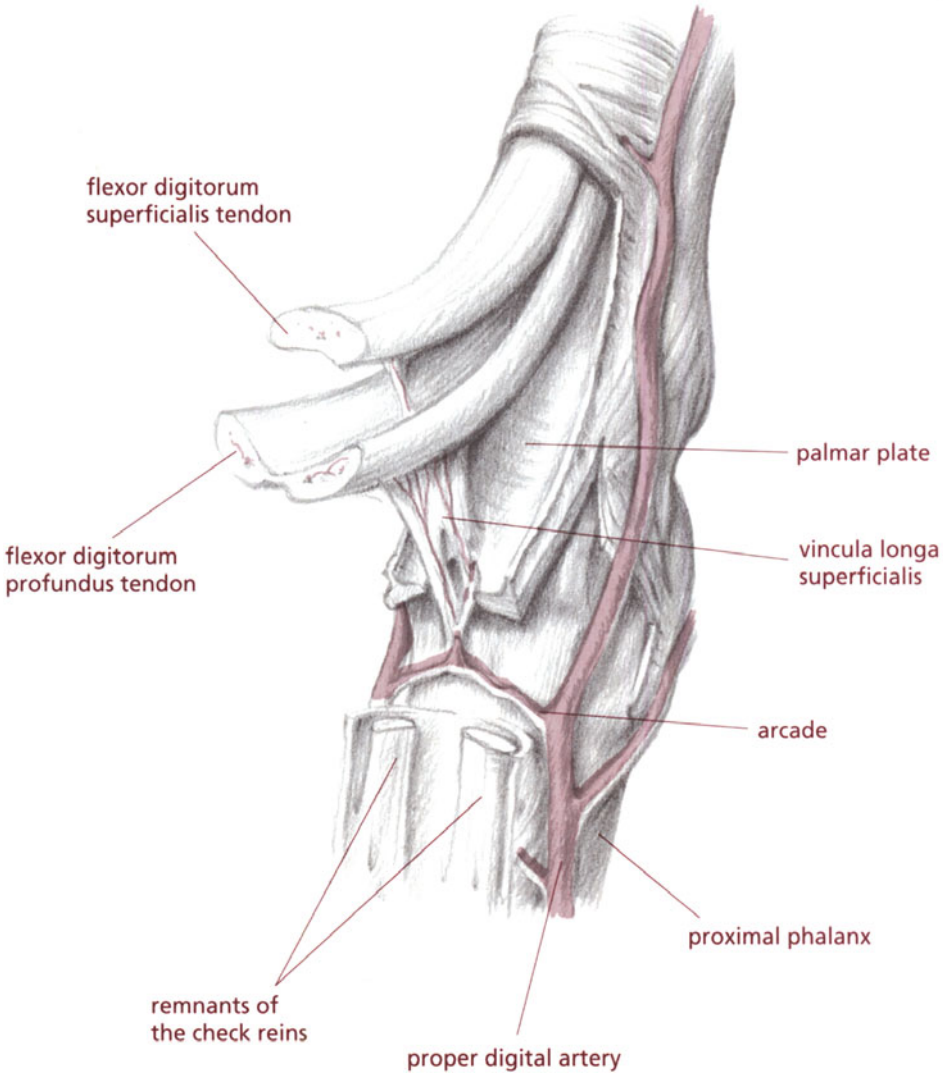

Fig. 55d. Gentle manual pressure on the proximal interphalangeal joint and flexion of the metacarpophalangeal joint usually relieve the secondary contracture. If there is persistent elastic resistance or a "snapping" effect, the shrunk accessory ligaments can be completely resected. The transverse, arterial arcade (communicating vessel) may neither be divided, nor disrupted by accident. Immediatly after the procedure, active exercises have to be started and a dynamic extension splint needs to be worn.

21.4 Corrective osteotomy

MOBERG (1973) recommended subcapital resection of the proximal phalanx to reduce flexion contracture of the proximal interphalangeal joint. The head of the proximal phalanx is then tilted by 90 degrees towards the palm. In this new position, it is attached to the metaphysis of the proximal phalanx by an osteosynthetic technique. This type of corrective osteotomy can bring an excessive flexion contracture into a functionally beneficial position (Fig. 56a–c). The dorsal wedge osteotomy suggested by TONKIN et al. (1985) represents a modified version of this procedure. By resecting a wedge-shaped segment (the dorsal cortex being the base), the initial extension deficit is reduced from 90 degrees to, for example, 30 degrees. With 60 degrees, the range of motion remains unchanged.

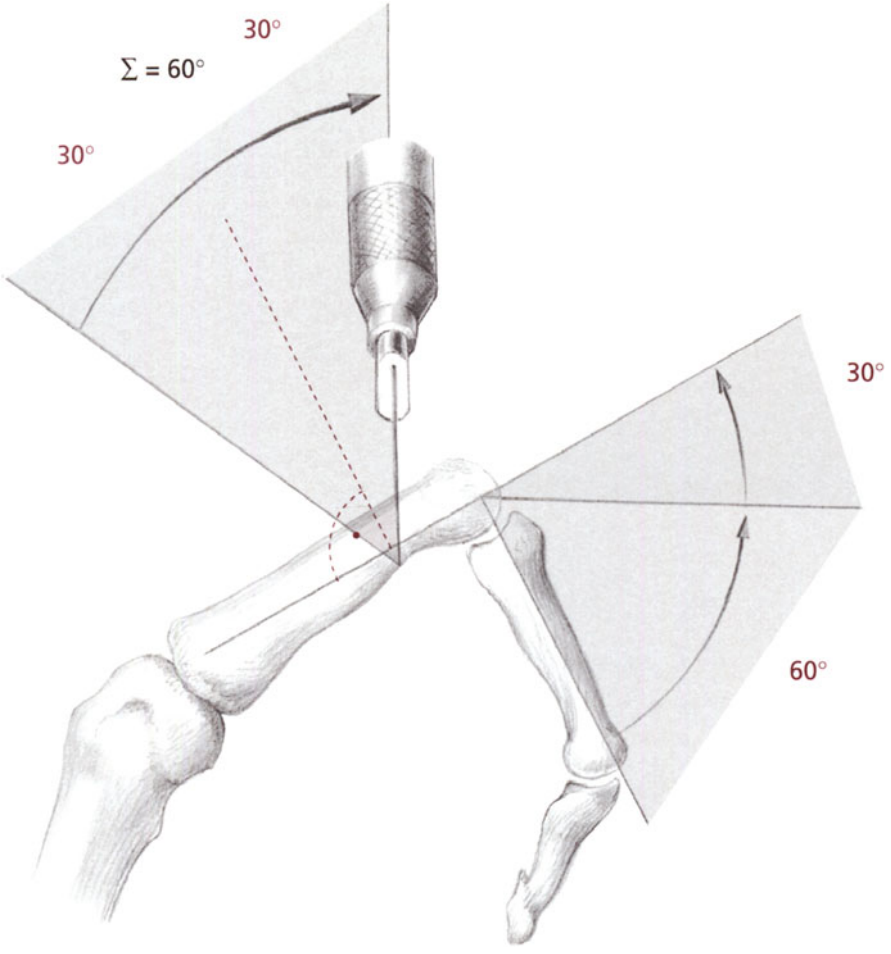

Fig. 56a. Corrective osteotomy modified by Moberg and Tonkin: subcapital wedge resection and osteosynthesis with the head of the proximal phalanx hyperextended. The dorsal angular distance is 60 degrees prior to closing wedge osteotomy. Reduction of the initial flexion contracture to about 30 degrees postoperatively.

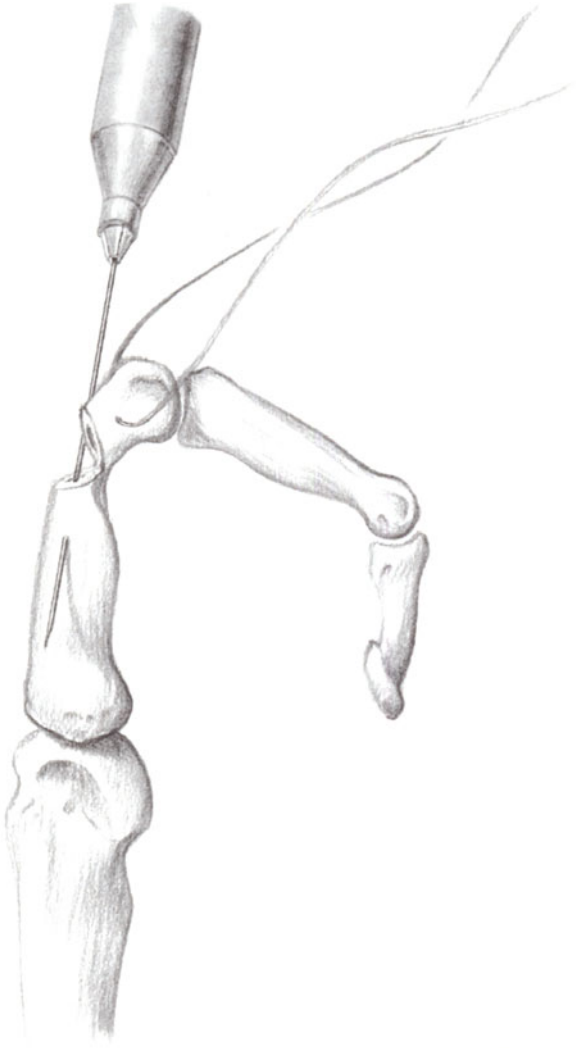

Fig. 56b. Fixation is achieved in combining diagonal K(irschner)-wire with looping intraosseous wiring. Vigorous functional exercise increase the compression.

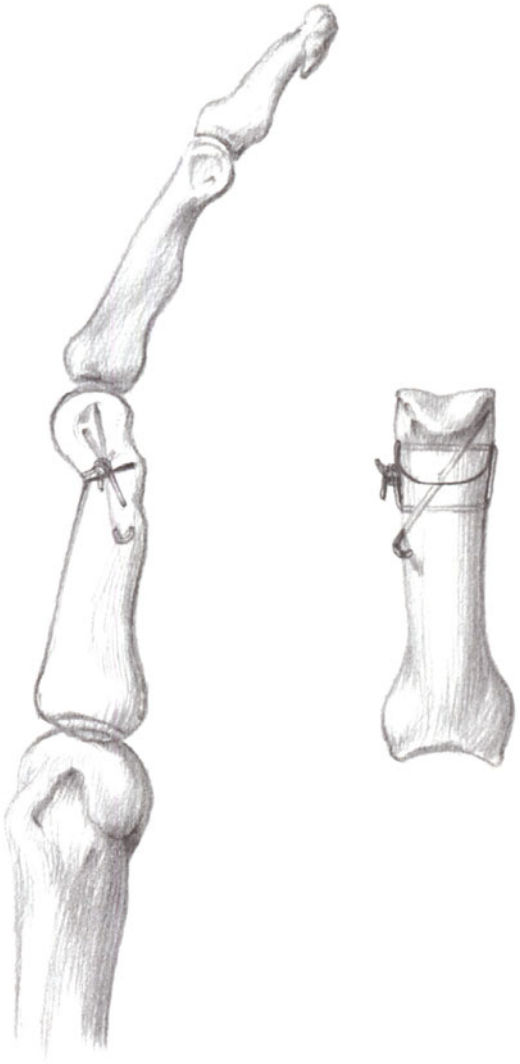

Fig. 56c. Executed closing wedge osteotomy. Reduction of the initial flexion contracture from approximately 90 degrees to about 30 degrees. Range of motion remains identical at 60 degrees.

21.5 Arthrodeses

Arthrodesis results in a stable, painless fusion of the joint and places the contracted finger in a more functional position, but has the disadvantage of stiffness.

In case of more than 90 degrees of contracture of the proximal interphalangeal joint, which cannot be resolved by any of the above methods, arthrodesis is indicated.

This is done by first exposing the proximal interphalangeal joint via a dorsal curvilinear incision centered over the joint. The central strip of common extensor tendon is isolated and folded up as a V with a distal base. After removal of the articular cartilage of the head of the proximal phalanx and the base of the middle phalanx, the "cup and cone" method is used to juxtapose the remnants of the PIP joint as needed. The "cup and cone" method allows final adjustments and an intimate bone-to-bone contact in three planes without any supplementary steps. The desired angle of fusion is held with two Kirschner wires drilled parallel to each other. Preferably dorsal to the mediolateral line, a bony channel is drilled transversally at the base of the middle phalanx approximately 4 mm from the former PIP joint. An intraosseous wire is pulled through this bony channel. A supplementary interosseus wire in a figure-of-eight pattern around the two K-wires completes the tension band arthrodesis, which is tightened to increase the compression. The ends of the Kirschner wires are bent and cut. The triangular V-shaped ligamentous flap is repositioned cover the hardware. Careful hemostasis and skin suture complete the procedure (PECHLANER et al., 1998; Fig. 56d–f).

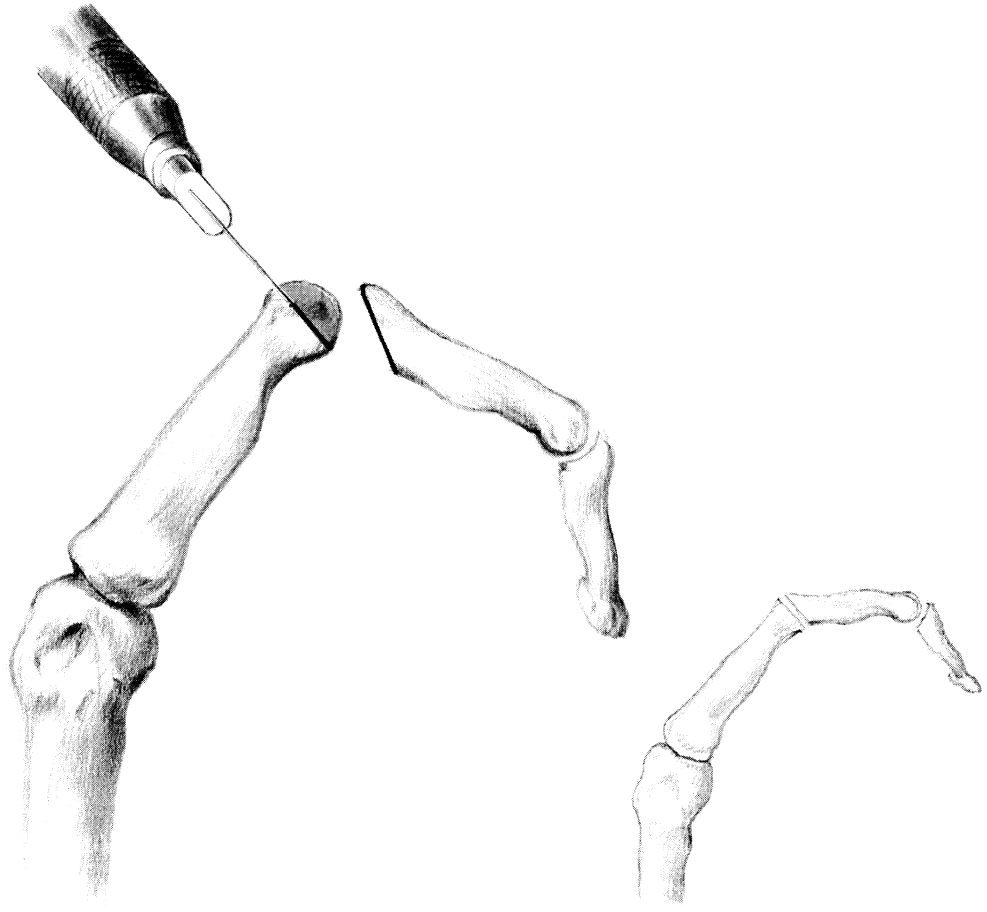

Fig. 56d. After exposing the PIP joint, the condyles' articular surface (head) of the proximal phalanx is resected, providing a large cancellous contract area for the arthrodesis. The fusion angle varies between 30 to 50 degrees. Correspondingly, the middle phalanx is resected as wedge form dorsal-proximal to palmar-distal. A transverse hole for the tension wire is drilled 4 mm distal to the osteotomy.

Fig. 56e, f. After reduction, correct angulation and rotation are varified. By means of two parallel Kirschner wires a temporary fixation is achieved. The tension band is placed around the K-wires in a figure-of-eight. The K-wires stabilize the arthrodesis longitudinally and the twisted tension wire provides intrafragmentary compression. Finally the protruding ends of the Kirschner pins are bent backwards, cut and buried in the bone or soft tissues, so that the tension wire cannot slip. When properly executed, this technique allows immediate aggressive rehabilitation: due to the tension band principle, active flexion will cause good alignment and progressive compression of the fusion gap.

As an alternative, a dorsal and straight longitudinal incision may be used to divide skin, subcutaneous tissue, plus parts of the capsular ligaments in one step. The articular cartilage of the head of the proximal phalanx is resected with a microsagittal oscillating saw at 90 degrees to the digital axis. The base of the medial phalanx is resected as a wedge in order to retain a functionally favorable angle. The two prepared surfaces allow any rotation to set the correct alignment for the desired fusion. Arthrodesis can be accomplished by either tension band arthrodesis, as described above, or one can combine one simple diagonal Kirschner wire with a transverse intraosseus tension wire, which forms a loop. Instead of one intraosseous loop, an extra loop on the contralateral side may provide more compression, but adds more bulk. Tightening of the tension wire can be increased by twisting the ends up to three times (Fig. 56c).

21.6 Amputation

Amputations concern mainly the little and finger and in exceptions the ring finger. Because fibrosis also affects the MP joint, exarticulation at that joint level should be considered. Filet flaps can be fashioned from the soft tissue of amputated fingers or from other areas and may be used to cover substantial defect of the palm (Fig. 57a, b).

The filet flap (cheiloplasty) is superior to skin grafts or any distant flap as it matches the typical texture and color of hand tissue, its skin is sturdy, and it provides unsurpassed sensibility (Koegel et al., 1995).

The on-top plasty can be used by experienced microsurgeons to avoid neurinomas. This transfer of the distal phalanx with its neurovascular pedicles avoids a stump at MP level, gains length, maintains locoregional sensory perception and increases the span of grasp in hands affected by Dupuytren's disease. The distal phalanx is dissected free and both neurovascular are preserved as pedicles. A segmental amputation of the afflicted finger is performed distal to the base of the proximal phalanx. The on-top plasty is transposed to the recipient site, where it is fused to the stump of the proximal phalanx (Baiback, 1990). One of the main advantages of on-top plasty is that there is no stump in spite of partial amputation.

Fig. 57a. Owing to functional impairment, for example as a result of multiple Dupuytren relapses or prolonged intraoperative ischemia, the little finger had to be amputated.

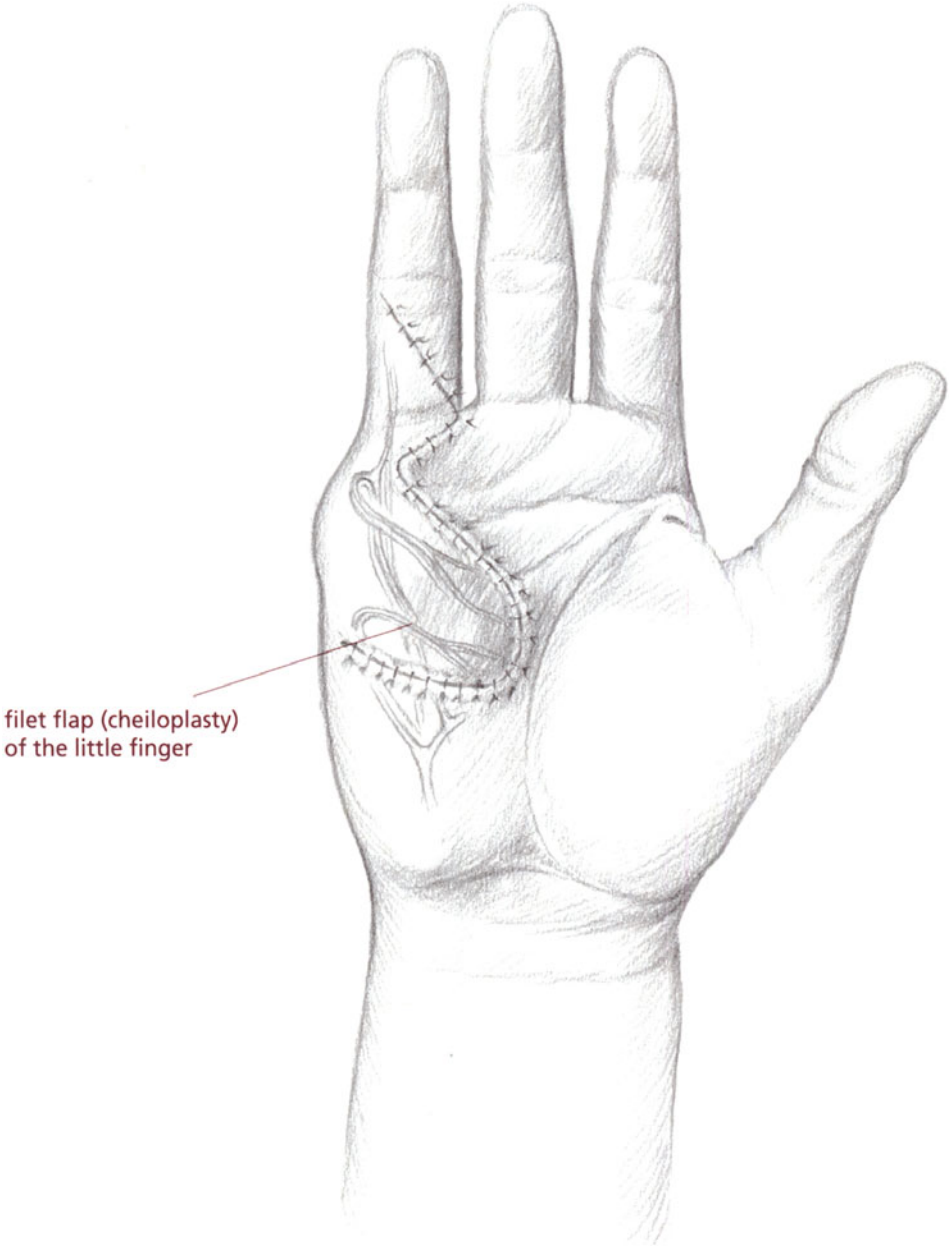

Fig. 57b. After exarticulation of the MP 5 joint, the soft tissue envelope, which incorporates the two neurovascular bundles, is used as a so-called filet flap (cheiloplasty) to cover the palmar defect.

22 Postoperative treatment

22.1 Follow up treatment after minimal invasive procedures

Wounds after fasciotomy, needle "fasciectomy," and segmental aponeurectomy are dressed with Sofratulle (or vaseline gauze) and a "conforming dressing". Physiotherapy should be started immediately after the procedure. Suction drains need to be removed on the second post-operative day (POD). The hand should be elevated to minimize edema, and antiphlogistic drugs (such as diclofenac) should be given. The range of motion should be increased under supervision of a physiotherapist. Sutures are removed on POD #10. Baby oil or vaseline should be applied to smooth the scars.

A night extension splint is absolutely essential for residual contractures.

22.2 Postoperative management after partial and total fasciectomy

After partial or total fasciectomy the hand should be dressed with Sofratulle gauze. This dressing should, in turn, be covered with a steel wool pack to absorb fluids and to exert gentle pressure to prevent fluid collections. The bandage should be a "conforming dressing", which means that a uniform amount of pressure is applied to the entire hand. In case of fasciectomy limited to the palm, the hand should be immobilized for five days with a plaster cast extending to the metacarpophalangeal joints. After digital dissection a plaster cast in intrinsic-plus position up to the distal phalanges is required.

Early and temporary abduction of the digit by the interosseus muscles serves to prevent edema. The limb should be suspended or rested on a wedge-shaped hand cushion.

The first dressing change and removal of the drain are due on the second POD. Drainage volumes of $>$ 50 ml in 24 hours and water cushion-like swellings of the palm may be the result of a hematoma. This condition requires operative revision.

Fibrin glue is beneficial in preventing recurring hematoma in cases of extensive dissection (Brenner, 2001). As shown by Eide et al. (1985) and Werber (1992), the fibrin glue sealant technique results in fewer necroses of the wound margins and, more importantly, fewer instances of palmar hematoma. Of the two-component fibrin glues, Beriplast® (Aventis) is considered safe because it does neither contain enveloped nor non-enveloped DNA and RNA viruses, owing to its pasteurization.

22.3 Specifics of the open palm technique

Forty-eight hours after the operation passive flexion and extension of the operated, "open," hand is first practiced under instruction. The first dressing change should be done after one week. "Controlled scar formation" (cicatrication dirigée) takes four to eight weeks altogether. During that time, the proximal interphalangeal joints should be rested on a static extension splint over night and on a dynamic extension splint during the day (Allieu et al., 1988).

22.4 Aftercare following skin grafts and local flaps

Skin grafts have to be checked on the second day to rule out hematomas, which might lead to graft loss. The bolster dressing should be kept on for at least five days after the operation. Grafts need a plaster splint reaching the fingertips; the intrinsic-plus position is ideal.

Local flaps have to be inspected on a daily basis for signs of compression, strangulation, or traction of the pedicle, which would necessitate an immediate revision.

22.5 Aims of physical and occupational therapy

Physiotherapy aims at achieving unrestricted range of motion and complete sensory recovery of the operated hand.

Fist closure and complete extension are unlikely in the early days. During the first three weeks the patients can barely manage to touch palm with the fingertips. Complete fist closure cannot be expected earlier than three months postoperatively and requires intensive exercise. Thermotherapy may be started after the fourth week. Physiotherapy takes about four to six weeks, after that the patients will have to follow an independent exercise routine (McFarlane, 1987).

Occupational therapy aims at training functional processes of the operated hand for domestic and professional use, if necessary by using aids (for example, a larger handle).

The ability to work is restored between six and eight weeks after the "standard" operation. The scar needs permanent care and can withstand "normal" mechanical strain only after three months (Van De Kerckhove, 2000).

Adhesive silicone sheets overlying the wound and compression bandages both help in the treatment of hypertrophic scars (Prosser et al., 1996).

In cases of prior capsulotomy, patients have to permanently wear a dynamic splint for at least six weeks, and at night for another six weeks (Crowley et al., 1999).

Patients will have to be informed in advance that Dupuytren's disease does not count as an occupational disease and does therefore not make them eligible for early retirement, despite its 15% coincidence with trauma of the same extremity (Brenner, 2002). The physician as well as the physio- and ergotherapists, who will treat the sequelae of the disease and guide the Dupuytren patient, will have to be aware of this fact.

Finally, early communication between the surgeon and the patient's physiotherapist via written orders or verbal exchange is inevitable to reach the aforementioned goals.

23 Errors, hazards, and complications

Hazards and complications begin with patient selection. Care has to be taken to strictly adhere to indications for and contraindications to fasciectomy.

Surgery during the early stages of the disease may cause complications. A removal of nodules is never indicated nor is surgery during Tubiana stage I and II. Only if the tabletop test is positive should fasciectomy be considered.

Disregard for the natural history of the disease may be hazardous. Fasciectomy during the nodular stage needs to be avoided as it may lead to an exacerbation. The same is true for an intervention for early cord formation.

For recurrences, surgery is only indicated in the presence of functional impairment.

The operative correction of digitopalmar contracture demands skill and expertise. A surgeon lacking profound experience in hand surgery should never be entrusted with a fasciectomy (Brenner, 2002).

Complications can occur at any point intra- or perioperatively. It is testimony to a surgeon's skill to recognize such problems immediately. The prompt and adequate management of complications as soon as they arise has a significant impact on the short- and long-term result (Boyer et al., 1999).

23.1 Hematomas

Hematomas induce an increased intrapalmar pressure, potentially followed by necrosis of marginally perfused flaps. Failure to evacuate a hematoma results in markedly limited range of motion. In addition there is a tendency for postoperative edema; this also explains a higher rate of reflex sympathetic dystrophy.

Removal of only a few stitches on the palm, spreading of the wound edges and attempting to relieve a hematoma at the bed-side are usually insufficient. A second anesthesia and revision in the operating room are imperative. The entire wound must be opened and the hematoma carefully removed. The wound requires irrigation and the source of bleeding must be determined. After meticulous hemostasis, drains are inserted. Immobilization with a plaster splint may be needed.

Should the area around the wound get inflamed or if there are systemic signs of infection (fever), a specific antibiotic therapy must be instituted.

23.2 Lesions of the neurovascular bundles

The frequency of iatrogenic neurovascular injuries can be decreased by the use of a tourniquet and surgical loupes, and, in cases of recurrence, by the obligatory use of an operating microscope.

Severed digital nerves lead to painful iatrogenic neuromas and sensory loss if they are not coapted immediately by epiperineural sutures or nerve grafts.

Loss of digital perfusion following fasciectomy and release of a contracted finger joint may occur due to vessel spasm, accidental separation of the digital artery or because of the acute straightening of the PIP joint. Intraoperative stretching of finger vessels, for example in cases of progressive contractures or those of long duration, may lead to inadequate perfusion, which can be transient or permanent.

The risk of unintentional division of a vessel can be decreased by prograde dissection using the aforementioned magnifying devices.

Detailed knowledge of the different cord formations makes it possible to predict the displacement of the neurovascular bundles to a certain extent. This lowers the risk of severing the bundles by accident (Boyer et al., 1999).

The application of warm saline-soaked gauze combined with local smooth-muscle relaxants (20% lidocaine or papaverine) and dextrane infusions are the best strategies to prevent vascular spasm.

During surgical revision for a relapse, fingers with only one digital artery remaining ("one-vessel digit") are prone to ischemia, when microvascular reconstruction of the vessel does not succeed. For this reason, preoperative angiography is mandatory, especially if a previous (unrepaired) vascular injury has been recorded. Without such precautions, an amputation (exarticulation) must be anticipated.

The risk of amputation because of intraoperative straightening can be diminished by incomplete extension of the finger during surgery (while the MP joints are flexed) or by resection arthrodesis of the contracted joint, which causes a beneficial shortening of the finger.

Preoperative passive stretching and splinting constitute other options to overcome residual contracture.

23.3 Necrosis of the wound margin

The incidence of necroses of the skin margins varies between 3.2% and 15% (Hueston, 1961; Stuhler et al., 1975). The lowest rate occurs in longitudinal incision with serial Z-plasties. Transverse incisions after McIndoe are associated with a necrosis rate of 4.2% plus a 10.6% risk of skin edge necrosis. This contrasts with L-shaped incisions, which have a 5.5% rate of manifest necroses and additionally an 11.2% rate of skin edge necroses (Stuhler et al., 1975).

The incidence of wound edge necroses can be diminished by proper design of the flaps, by paying attention to proper circulation, by early evacuation of seromas and hematomas, and by avoiding strangulating stitches (Brenner, 2002).

The consequences of necroses are deep infections, bare neurovascular bundles, or a denuded flexor tendon sheath, or even an exposed joint after arthrolysis or protrusion of implants after arthrodesis.

Deep infections must be treated with scrupulous debridement, swabs for culture and sensitivity, insertion of drains, and antibiotic therapy. Loose implants have to be removed; skeletal stabilization is achieved either with external fixators or a plaster splint. Soft tissue losses must be covered by flaps as early as possible.

23.4 Graft failure

The loss of skin grafts is due to faulty assessment of the recipient site, the selection of an inappropriate skin graft, inappropriate graft size (tent-like lift-off from the recipient site). Subcutaneous oozing and insufficient immobilization of the extremity prevent capillary ingrowth into the graft.

A tie-over dressing to the skin guarantees an intimate contact of the skin graft and prevents movement, which may impede healing. A plaster splint worn for approximately 10 days helps with the intended immobilization.

If graft failure occurs in spite of these precautions, the properly conditioned wound has to be resurfaced by a second graft or flap, be it homo- or heterodigital. An alternative will be healing by secondary intention; this will inevitably lead to an unpleasing scar contracture and thus decrease hand function.

23.5 Flap necrosis

Flap necroses can be avoided by proper flap design with respect to the length-width ratio. A ratio greater than 1:2 is likely to suffer from insufficient vascular supply. Any traction on the wound edges is undesirable. Meticulous hemostasis prevents accumulation of seromas and hematomas underneath the flap. When we apply dressings we avoid any exaggerated external compression and strangulation.

Staying in the correct plane is important for flap dissection.

Accidental buttonhole-like skin perforations may also interfere with perfusion and cause flap necrosis (Boyer et al., 1999; McGrouther, 1999).

Any hope or expectation that tissue with questionable viability will heal spontaneously is in vain. If the skin does not appear pink after a waiting period of 10 minutes or application of warm, moist compresses, or if bluish discoloration from venous congestion persists, the tissue should be resected and the defect covered according to the above-mentioned principles (Brenner, 2002).

23.6 Reflex sympathetic dystrophy (algodystrophy)

The consequences of Sudeck's dystrophy are disturbed pain perception, abnormal sudomotor function (sweating, discoloration), trophic and secretory changes (edema, accelerated nail growth, hypertrichosis), a frozen hand with diffuse osteopenia, subchondral lucencies (erosion or cysts) and juxtacortical demineralization of all bones of the hand. Preventively, fasciectomy should not be combined with other major hand surgical procedures. An atraumatic surgical dissection technique that minimizes segmental devascularisation is deemed to lower the incidence of reflex sympathetic dystrophy. The treatment is always matched to the disease stage, and other disciplines are involved in the therapy (the surgeon, pain clinic, physiotherapist, and, if needed, a psychiatrist; [Koman et al., 2000]). The goal of this treatment is to eliminate pain and to improve the local blood supply through blocks (stellate ganglion block, continuous epidural block) or pharmacologically (alpha 1 and alpha 2 blocking agents, anticonvulsive drugs, competitive inhibitors of neurotransmitters, etc.), acupuncture, biofeedback. The range of motion ("stress loading") should also be increased without causing pain and under supervision of and instruction from a physiotherapist.

24 Results

24.1 Functional results and recurrence rates

While the postoperative range of motion of the metacarpophalangeal joint is not so much determined by variables such as familial history, age at onset, preoperative joint status, type of operation, duration of surgery, and level of surgeon's experience, Legge et al. (1980) proved convincingly that the interphalangeal joint range of motion can be predicted on the basis of anatomical factors, such as the number of joints involved and the preoperative state of contraction. The postoperative range of motion of the little finger is, however, determined by the primary contraction status of the MP 5 joint, because of the metacarpophalangeal assemblage nucleus located here (Zancolli et al., 1992). The regained range of motion of the little finger can thus serve as a parameter for a "successful" operation in Dupuytren's disease.

Additionally, Adam and Loynes (1992) showed that primary and secondary fasciectomy lead to a statistically significant postoperative range of motion (ROM) improvement of the metacarpophalangeal joints compared to the interphalangeal joints. This holds true for static measurements and for the prognosis.

Table 2 presents an overall summary of manual function, listed by type of fasciectomy and assessed with various grading methods.

Opinions vary with respect to the point in time, which allows the most reliable prediction of recurrence rates.

Hueston (1963), for example, reported that in his patients 87% of all recurrences occurred during the first two postoperative years. Millesi (1965, 1981) in contrast related from his comprehensive records that 48% of the relapses occurred more than three years after the initial procedure.

Tubiana and Leclercq (1986) confirm that only 34% of relapses occurred within the first two years after surgery, but during a five year follow-up they noted a relapse rate of 50%, and after 10 years the relapse rate had risen to 65%. But 16% of relapses took a relatively asymptomatic clinical course, and only few patients needed another operation (Ebelin et al., 1987).

Forgon et al. (1988) documented that partial fasciectomy (n = 95 procedures) initially improved the ROM by 41%. Later, however, manual function became clearly impaired because of an 18% relapse rate within the first five postoperative years. The authors observed a relative stagnation of the recurrences in years two and three. The late functional results after five years showed a further improvement in ROM of 22.6% compared with the status before surgery.

Huber (1987) reported similar experiences from Berlin.

After more than 10 years, Norotte et al. (1988, 1989) were able to reexamine only 20% (n = 58) of the Dupuytren patients who had had a fasciectomy and standard postoperative regimen. The overall recurrence rate was 71%. 63% of the recurrences appeared within the first two years. Between the second and fifth year after surgery 32% had relapses. Only another 5% of relapses were diagnosed after this period. Retrospectively, the authors summarize the worst relapse factors. In descending order of importance, these are: (1) age less than 50 years; (2) clinically advanced stage of contracture corresponding to stage III or IV at the time of the initial surgery. (3) In the relapse group, the proportion of patients who had epilepsy, misused alcohol, had ectopic fibromatoses, or had a familial diathesis varied between 75% and 92%. (4) The authors believe that the recurrence rate is not dependent on the surgical technique.

Honner et al. (1971), who performed fasciectomies on 138 hands in Australia (3/4 total, 1/4 par-

tial fasciectomy), report a recurrence rate of 41%. They state that the disease expands in 20%.

In a 5-year follow-up of total fasciectomies, MILLESI (1981) documented that disease progression and recurrence were definitely less aggressive after this procedure. He therefore advocates total fasciectomy. Experienced surgeons can reduce the increased complication rates secondary to the extensive dissection in total fasciectomy to rates comparable to partial fasciectomy. According to SENNWALD (1990) complications are directly linked to the skill of the surgeon.

Relapses and, occasionally, progression of Dupuytren's disease are particularly frequent in the first web space. The causes for this are the three fibrous cords—the pretendinous cord to the thumb, the proximal transverse commissural cords and the natatory cord (involved distal transverse commissural band GRAPOW)—which often go unnoticed (TUBIANA and DEFRENNE, 1976).

After a follow-up period of at least one year (36 months average, seven years maximum) after using the open palm technique, ALLIEU and TEISSIER (1987) report a recurrence rate of 23/77 (29.7%) and a Dupuytren progression of 15/77 (19.4%) in the non-operated area. This study encompassed 77 out of 164 hands.

Out of a total of 868 open palm procedures, FOUCHER et al. (1992) reexamined 107 patients with 140 such procedures on fingers and a follow-up of at least five years. Their relapse rate was 40.6%, 23% of which needed a second intervention. The extension rate for Dupuytren's disease was 39% in the same group.

At a mean follow-up period of 36 months, TONKIN et al. (1984) report a relapse rate of 4% inside the full thickness grafts and of 33–42% outside the grafts in 41/100 patients treated with dermatofasciectomy.

BROTHERSON et al. (1994) did not find any signs of relapse underneath the full thickness grafts in 34 British patients who had dermatofasciectomies performed by a senior surgeon, and who were followed for an average of 100 months (80–120 months). In none of the cases did the digitopalmar flexion contracture reach more than 15 degrees.

MOERMANS (1996) performed minimal invasive segmental aponeurectomies in 59% (n = 141/240) on 173/292 hands, which he reexamined 2.9 years after the procedure. He reports an extension of Dupuytren's disease in 16% (n = 27) and relapses in 23% (n = 39), and a combination of both in 15% (n = 26).

FOUCHER et al. (1998) reviewed 171 patients treated by percutaneous needle fasciotomy, 52 of whom had complications. This is particularly remarkable because the "surgeons" modified the percutaneous needle fasciotomy procedure and mostly concentrated on the palm. The short-term improvement was 72.1% (median preoperative extension deficit 46.7%, median postoperative extension deficit 13%). As expected, the metacarpophalangeal joints had a better ROM outcome (79.6%) than the interphalangeal joints (53.7%). In one group comprising 65 hands, which were reexamined after an average of 2.5 years, seven hands had improved by a mean ROM of 14 degrees, whereas 35 hands had improved by 29 degrees.

The overall extension (progression) of digitopalmar fibromatosis in the group of patients treated by needle fasciotomy was 54%.

Table 3 summarizes some of the rates of progression and relapse in Dupuytren's disease as cited in the literature.

24.2 Complications depending on the surgical technique

A comparison of results only on the basis of regained range of motion, progression and relapse rates remains incomplete if the inherent complications of the procedures are not analyzed. In conclusion, we will explore such problems.

The most frequent and also most feared intraoperative mishaps include damage to neurovascular structures and problems with soft tissue perfusion. Impaired circulation can occur because of uni- or bilaterally severed digital arteries, because of external vascular compression, or because of constriction by surrounding fibrotic tissue.

The number of nerve injuries during segmental aponeurectomies is reported as 2% (MOERMANS, 1991).

MCFARLANE and MCGROUTHER (1990) report an incidence of 0.8% (n = 1,339 operations) of divided digital arteries and a 0.1% incidence of necrosis related to the fasciectomies.

In an unselected cohort study on Dupuytren's disease, SENNWALD (1990) reports neurovascular damage in 17.5% (eight nerves, 10 digital arteries; n = 103) despite the use of surgical loupes. Look-

Table 2. Manual function

	Method	Assessment method	Postoperative result
Badois [1993]	Needle "fasciotomy"	Own system	"Good/poor" stage I: 91.9%/8.1%, "Good/poor" stage II: 88.6%/11.4%, "Good/poor" stage III: 83.3%/16.7%, "Good/poor" stage IV: 47.6%/52.4%.
Hueston [1961]	Partial fasciectomy	Own system	"Functional recovery" (in former job): 84.5%
McFarlane [1990]	Partial fasciectomy	Own system with ROM (= range of motion)	MP5-joint: "Perfect" 84% hand (finger:84%), PIP5-joint: "Perfect" 18% hand (23% finger), ROM increase: hand 41 degrees (finger 44 degrees)
De Maglio [1996]	Partial fasciectomy	Own system: "good" Deficit < 15 degrees, "satisfactory" deficit > 15 degrees, "poor" no improvement	"excellent" 55.2%, "good" 33.8%, "sufficient" 5.5%, "poor" 5.5%
McFarlane [1990]	Total fasciectomy	Own system with ROM	MP5 joint: "Perfect" 89% hand (finger 89%), PIP5 joint: "Perfect" 25% hand (13% finger), Increase ROM: hand 46.5 degrees (finger 42.1 degrees)
Brenner [1994]	Total fasciectomy	"Coefficient of improvement" according to Tubiana	70–86% ulnar type 56–88% radial type
Schneider [1986]	Open palm technique	Own system with ROM ("total active motion")	Improvement: in 88% of cases (D2:3 degrees, D3:11 degrees, D4: 32 degrees, D5: 31 degrees.
Shaw [1996]	Open palm technique	Strickland + Gloglovac- Method	Total ROM preoperatively: 48%, postoperatively: 96%
Hall [1997]	Dermatofasciectomy	Own system with ROM	65.7% PIP flexion > 85 degrees. 14.4% maximum 70 degrees achievable. Residual PIP contractures < 20 degrees: 76.5%. Residual contractures on PIP > 20 degrees: 27.9%

Tabelle 3. Progression and relapses in Dupuytren's disease

	Method	Period	Number (n)	Progression (%)	Relapse (%)
Luck [1959]	Fasciotomy	?	?	?	71
Millesi [1965]	Fasciotomy	3–5 years	n = 9	?	77.7
Lermusiaux [1997]	Needle fasciotomy	?	n = 799/3, 736 rays	?	50
Moermans [1996]	Segmental aponeurectomy	2.9 years	n = 141/240	16	23
Hueston [1961]	Partial fasciectomy	2 years	n = 96	28.1	12.5
Millesi [1965 +1981]	Partial or total fasciectomy	>5 years (1–12 years)	n = 577	Jointly	39
De Maglio [1996]	Partial fasciectomy	33 months (6–59 months)	n = 124	26.2	24.1
Norotte [1988]	Partial fasciectomy	10 years	n = 58	?	71
Leclercq [1994]	Partial fasciectomy	10 years (8–14)	n = 50/89	?	66
McGrouther [1999]	Partial fasciectomy	5 years (max. 20 years)	n = 100	?	50
Sennwald [1990]	Total fasciectomy	3–6 months	n = 103/256	?	21.4
Brenner [1994]	Total fasciectomy	47 months	n = 48/239	Jointly	39.7
Schneider [1986]	Open palm technique	5 years	n = 49	48	32
Allieu [1987]	Open palm-technique	36 months (1–7 years)	n = 77/164	19.4	29.7
Foucher [1992]	Open palm technique	Minimum *5* years	n = 107/140 procedures	39.0	40.6
Tonkin [1984]	Dermatofasciectomy	*36* months	n = 41/100	?	4% underneath skin graft, 33–42% under original skin
Ketchum [1987]	Dermato-fasciectomy	3.9 years	n = 24/36 hands	8	0
Brotherson [1994]	Dermato-fasciectomy	100 months (80–120)	?	?	0
Hall [1997]	Dermato-fasciectomy	48.4 months (24–100)	n = 67/90 radii	?	8

ing exclusively at primary interventions he reports neurovascular complications in only 3.9% (n = 3/77). All accidental injuries to digital neurovascular bundles occurred in TUBIANA's stage III of Dupuytren's disease. The remaining severed vessels or nerves (n = 15) occurred during reoperations and always in stages III and IV.

GELDMACHER (1994) reports an overall complication rate of 15.09% for 2,160 fasciectomies, among them 91% (n = 1,965) partial aponeurectomies, which were performed since 1956. These include 2.82% (n = 61) nerve lesions, 1.30% (n = 28) secondary amputations as a result of malperfusion because of divided arteries, 0.23% (n = 5) tendon injuries, 1.16% (n = 25) secondary hemorrhages, 2.18% (n = 61) cases of reflex sympathetic dystrophy, 2.68% (n = 58) infections, and finally 4.72% (n = 102) skin necroses.

In 566 patients with Dupuytren's disease from northern Germany (Lower Saxony), most of which were operated and followed by us (n = 383 partial fasciectomies, n = 126 total fasciectomies), the incidence of neurovascular lesions was 1.4% (2.8%) after partial fasciectomies (proportions for total fasciectomies in parentheses). The frequency of postoperative hematomas was 2.35% (4.8%). Infections occurred in 0.5% (0.8%) and delayed wound healing in 4.2% (6.34%). These data relate to the number of operated hands and not, as often seen in the literature, to the number of finger rays or the number of neurovascular bundles (BRENNER, 1994; 2002).

HUESTON (1961) believes that most of the complications are related to the extensive palmar wound, rather than the digital dissection, and, in particular, to the radical palmar resection usually claimed to present a "total" fasciectomy.

HOET et al. (1988) consistently relate the number perioperative complications to the number of finger rays with fibrotic involvement. 88% of those operated in the study group had a partial fasciectomy and the others a total fasciectomy. The series included 201 patients with 326 diseased hands. In addition to six accidentally severed digital arteries, 17 severed digital nerves were found. The authors cite nine cases of moderate infection and two cases of severe infection. The proportion of superficial to deep skin necroses was 44/7. The latter needed five skin grafts. Three cases of hematoma were treated conservatively, while 16 patients required revisions for hematomas. Wound dehiscence after suture removal was recorded in two patients after surgical treatment of Dupuytren's disease.

LUBAHN et al. (1984) compared postoperative complications after "closed" total fasciectomy (n = 115) with the open palm technique with transverse incisions (n = 38). The closed technique had a complication rate of 19% as opposed to 8% with the open palm technique.

Total duration of treatment being equal, FOUCHER (1985) and WULLE (1991) found that the open palm technique has the lowest rate of postoperative complications.

Among 107 patients with 140 open palm interventions, FOUCHER et al. (1992) observed nerve lesions in 3.1% and reflex sympathetic dystrophy in 7%. Although 3.5% of these patients had been anticoagulated, the rate of hematomas requiring revision was only 0.7%.

ALLIEU and TEISSIER (1986) report a 3.6% incidence of nerve lesions with the open palm technique. The wound dehiscence (unrelated to hematomas) rate is 20.6% (n = 16/77). These authors report an 11.6% rate of algodystrophy and an incidence of "pre-algodystrophy" with persisting edema of 18.1% (n = 14/77).

Table 4 summarizes the complications associated with specific techniques.

Table 4. Complications associated with specific techniques

	Method (= cases)	Hematoma (%)	Neurovascular lesions (%)	Infection (%)	Necrosis (%)	Reflex sympathetic dystrophy (%)
Badois [1993]	Needle "fasciotomy" (n = 90/123 hands)	2.0	2.0	16 (skin tears and fissures)	?	
Lermusiaux [1997]	Needle "fasciotomy" (n = 799/3.736 rays)	0.8	5.2	?	?	1.8
Millesi [1965]	"limited resection" = Segmental resection (n = 31)	3.2	?	0	?	3.3 ("persistent edema")
Moermans [1996]	Segmental aponeurectomy	2.28	1.14	?	0.57	1.14
Millesi [1965]	Partial fasciectomy (n = 129)	3.1	?	?	6.2	4.65 ("persistent edema")
Norotte [1988]	Partial fasciectomy (n = 58)	?	3,4	?	25.8	3,4
Brenner [1994]	Partial fasciectomy (n = 383)	2.35	1,4	0.5	4.2	?
Geldmacher [1972]	Partial fasciectomy (n = 1565)	1.16	Nerves 2.82 Arteries 1.3	2.68	4.27	2.18
Millesi [1965]	Partial fasciectomy (n = 91)	7.7	?	?	12.1	5.5 ("persistent edema")
Sennwald [1990]	Partial fasciectomy (n = 103)	2.91	Nerves 9.7 Vessels 9,7	?	0.97	17.5
Brenner [1994]	Partial fasciectomy (n = 126)	4.8	2.8	0.8	6.34	?
Noble [1992]	Open palm technique (n = 75)	?	18.75 ("Hypesthesia")	6.25	5	?
Schneider [1986]	Open palm technique (n = 49)	0	?	0	0	12
Shaw [1996]	Open palm technique (n = 25)	?	4	?	?	4
Hall [1997]	Dermato-fasciectomy (n = 67/90 finger rays)	?	1.5	?	1.5 (amputations 1.5)	0

25 Conclusions

Observations on Dupuytren's disease tend to show that the disease occurs predominantly in countries with small homogeneous populations and spreads mostly inside one ethnic group. Because of the autosomal dominant transmission with high penetrance, mainly men in northern regions are afflicted, often those of Celtic or Viking descent (Saar et al., 2000).

About 1,500-50 BC many Celtic tribes had a highly developed culture that was extinguished by the Romans (McFarlane, 1997). The prevalence of Dupuytren's disease is therefore low in Mediterranean countries.

The disease prevalence has a pronounced north/south gradient. This epidemiological observation becomes quite obvious in large countries, such as France, which has access to the Atlantic Ocean as well as to the Mediterranean.

Brouet (1986) demonstrated a striking finding concerning the prevalence of Dupuytren's contracture among the French population. Owing to William the Conqueror's brother invading Sicily and the Mediterranean parts of France in 1,066 AD, the city of Toulon has a blue-eyed population with northern ancestors on the one hand. Dupuytren's disease occurs frequently in this population. The dark-eyed Toulonese population, which has Roman roots, has a much lower incidence of Dupuytren's disease. The Mediterranean city of Toulon, situated in the extreme south east of France, is regarded as an epidemiological enclave. It is known for the highest disease prevalence, which is 17%. Comparable, but slightly lower rates are reported for Normandy and Brittany, which were also occupied by northern invaders.

The worldwide incidence of Dupuytren's disease varies greatly. In the southern Mediterranean and Middle East, fibromatosis is almost unknown. In Asians , pure Afro-Americans or Black Africans, and among Gypsies or Indians and aboriginal Sami (Lapps), flexion contracture is believed to be rare and occurs only in individual cases (Brenner et al., 1994; Finsen et al., 2002).

These cases can be explained only partly on the basis of spontaneous genetic mutations. The incidence of Dupuytren's disease in the Japanese region of Osaka and Kobe is, for example, similar to that observed in the north European population. This is probably the result of ethnic migration (Egawa, 1985).

Cavalli-Sforza (1981, 1990) undertook linguistic studies and DNA analyses in that Japanese region and showed that the majority of the resident local population originates from the Urals, explaining the diathesis for Dupuytren's disease as a phylogenetic one.

It seems that advanced age (associated with occlusion of macro- and microvessels), genetic (Viking ancestry and familial diathesis), endogenous (diabetes mellitus with advanced microangiopathy), and environmental factors (nicotine abuse, alcohol consumption, diet rich in fat in northern countries) cause progressive luminal occlusion of microvessels. This induces local ischemia, the basis for the production and release of various oxygen free radicals (Murrell et al., 1992, 1994). Radicals cause fibroblast proliferation, perpetuating local ischemia. The cellular density of fibroblasts suppresses type III collagen synthesis, shifting the ratio of type I to type III collagen.

In our recent analysis of the extracellular matrix distribution and concentration of glycosaminoglycans in digitopalmar fascia affected by Dupuytren's disease, we noted increased resinification caused by an increase in sulfated glycosaminoglycans in the digitopalmar continuum (dermatan-4 and dermatan-6 sulfate) that occurred in the clinically striking cord formations. Additionally, we

found that the radial type of Dupuytren's disease is actually an expansive ulnar type and not an independent entity.

Simultaneous determination of plasma GAG disaccharides in patients with Dupuytren's disease of TUBIANA stage IV proved that the quantitative increase in the total amount of detectable glycosaminoglycans was about 4.2-fold higher than in normal controls. The concentration in control subjects was 9.3 compared to 39.1 µmol/l in patients with Dupuytren's disease. This difference was highly significant ($p < 0.001$). Among all glycosaminoglycans, hyaluronan showed the most significant increase by a factor of 8. These data indicate a continuous remodeling and maintenance process of the diseased palmar fascia. The chondroitin level was 6.1 times higher in Dupuytren's patients than in controls (5.1 µmol/l vs. 31.1 µmol/l; $p < 0.001$).

In patients with Dupuytren's disease, the concentration of chondroitin-4 sulfate, the substance produced in the next biosynthetic step, was increased 1.8-fold , i.e. from 2.6 µmol/l (normal level) to 4.8 µmol/l,. The most noticeable finding is that dermatan-4 sulfate and dermatan-6 sulfate are not detectable (nanomol range) in plasma of patients with Dupuytren's disease because an intracellular sulphatase (uronosyl-5-epimerase) breaks down these substances inside the palmar fascia, preventing their release into the blood.

Clinical rather than serological parameters are obviously still the main guidelines for establishing the diagnosis of Dupuytren's disease.

But it is possible to predict stage IV flexion contracture with a 91.7 percent probability by discriminant analysis of the following eight plasma GAG concentrations: hyaluronic acid, chondroitin, chondroitin-4-sulfate, chondroitin-6-sulfate, dermatan, dermatan-4 and dermatan-6 sulfate, and uronic acid. The diagnostic accuracy of 91.7% does not require the patient to be seen or examined.

It thus becomes obvious that Dupuytren's disease is truly a systemic connective tissue disease with an ongoing remodeling process indicated by quantitative and qualitative changes of glycosaminoglycan concentrations. We hope that glycosaminoglycan determination will in the future become as important for the diagnosis of Dupuytren's disease or its diathesis as the rheumatoid factor test is today for other systemic connective tissue diseases. With current methods, the accurate and precise determination of plasma GAG concentrations takes two days.

Ectopic deposits are well known as Peyronie's and Ledderhose's disease as well as Garrod's knuckle pads. Such remote pleiotropic fibrosis can now be explained for the first time as the result of changing GAG plasma concentrations or by an altered GAG distribution in connective tissue. Moreover, all aforementioned locations are also characterized by a connective tissue fiber microarchitecture similar to that of the digitopalmar continuum. This explains the histologic uniformity of the wide-spread ectopic deposits (BRENNER, 1994; BRENNER et al., 2001; BRENNER, 2002).

Apart from being useful in diagnosing Dupuytren's disease, plasma glycosaminoglycan concentrations can also be a useful tool for monitoring patients at risk for progressive digitopalmar fibromatosis, for controlled trials, or for quantifying the response to any non-invasive treatment. The test may possibly also play a role in the assessment of the systemic effect of fasciectomies.

Certainly, Dupuytren's disease can develop, no matter if the palmaris longus tendon is present or aplastic (RAYAN, 1999).

We agree with MILLESI's "internal theory" (1981, 1992). According to this theory, the disease affects primarily the microarchitecture of the preexisting connective tissue structures, including the digitopalmar continuum, digital fascia, and the transverse, longitudinal and vertical septa. Fibroblast rich nodules represent the initial stage. Eventually, central, spiral, longitudinal, and natatory cords form during the final stage. The initial nodular stage, which is histologically distinct because of its high density of fibroblasts, matures and gives way to an almost acellular cord stage. It is therefore useful to distinguish these stages histologically: proliferative, involutional, and residual stage (MAGRO et al., 1996, 1997).

Fibroblast proliferation within the three-dimensional connective tissue architecture results in adhesions, which block manual function.

In a strictly clinical and descriptive sense, it may therefore still be justified to distinguish between the radial and ulnar types of Dupuytren's disease, even if these are not independent entities.

Of all the clinical classifications, the TUBIANA staging scheme seems particularly appropriate to describe extension and abduction deficits.

With regard to interventions, more partial than "radical" fasciectomies are now performed. Minimal invasive traditional procedures, such as fasciotomy and segmental aponeurectomy are becoming more popular. These efforts have culminated in the experimental endeavor to remove the diseased fascial tissue through an endoscope.

Innovative methods include extension, respectively elongation techniques and their various modifications, such as those of MESSINA (1994), CITRON et al. (1988), or PIZA-KATZER (2000). All these have the need for a two-stage fasciectomy or at least fasciotomy in common.

Therefore, fasciotomy is seeing kind of a renaissance, both as a supplementary technique and as percutaneous needle fasciotomy.

On the other side of the spectrum, we find ultraradical procedures such as dermatofasciectomy with full thickness grafts, which assume the role of a "firebreak" within the complete range of treatments for recurrences.

It is essential to underline that these different surgical techniques are not competing with each other, but that each single one of them has specific indications.

The following methods have these advantages:

Fasciotomy is a minimal invasive and quick procedure that can be performed in outpatients under local/regional anesthesia. Even very ill patients (e.g. nursing home patients in poor overall health) may be candidates for a fasciotomy.

Needle "fasciectomy" (really a fasciotomy) is also speedy, minimally invasive, and can be performed under local/regional anesthesia and as an outpatient procedure. It is also suitable for patients with rheumatoid arthritis who are being treated with cortisone, and it enables patients to move their hand immediately. The fact that physicians who have not had any surgical training can perform this procedure is not necessarily an advantage.

Segmental aponeurectomy can be performed as an outpatient procedure in patients of any age. After local/regional anesthesia, limited undermining of the skin, and regional removal of the fibromatous tissue, prompt improvement can be expected. That makes it possible to start physiotherapy immediately.

Partial fasciectomy can be performed in younger and older patients. Simultaneously, minor hand surgical interventions (such as anular ligament release) can be performed. Partial fasciectomy is a technique favored for treating relapses. Its complication rate is low, owing to good exposure. Partial fasciectomy is done as an outpatient or same-day surgery procedure under brachial plexus block. The short period of immobilization, which allows physiotherapy to start early, is an additional advantage.

Total fasciectomy is advantageous, particularly in the expanding (widespread) ulnar type of Dupuytren's disease or in the so-called radial type, and it can be used in patients with multidigital contractures. Total fasciectomy also reaches the first web space. In addition, it allows the removal of vertical septa and natatory ligaments. The perfect visualization of all-important anatomical landmarks during this procedure has to be emphasized. If there is extension into the digits, we will try to maintain skin "bridges" between the distal palm and the base of the proximal phalanx.

It is also possible to combine total fasciectomy with minor simultaneous hand surgical interventions.

A gratifying element of open palm technique is that one can dispense with skin grafts or flaps. Patients can be sent home early. Low postoperative morbidity is another argument in favor of this procedure.

In our opinion, dermatofasciectomy offers the ultimate solution. Excised fibromatous areas covered with full thickness grafts act as "firebreaks" in digitopalmar flexion contracture. This procedure offers good protection against relapses.

The special disadvantages of the individual procedures are as follows:

Fasciotomy entails a great risk of injuring neurovascular bundles and tendons. Efforts at moving the finger early under physiotherapeutic instruction, will often lead to wound dehiscences with subsequent healing by secondary intention. Also, the rate of relapse is known to be high.

Needle "fasciectomy" is a "blind" type of manipulation with a high risk of iatrogenic injuries and a high rate of relapses. DUTHIE et al. (1997) reported that 66% of all patients treated with percutaneous fasciotomy needed a two-stage classical "radical fasciectomy."

Segmental aponeurectomy is often hampered by poor visualization.

Partial fasciectomy, however, often requires many reinterventions.

Total fasciectomy was disappointing with respect to how "radical" it really is. Wide undermining increases the risk of hematoma or necrosis of the dissected flaps. The high rate of reflex sympathetic dystrophy is another disadvantage.

The open palm technique is problematic because of the need for continuous physiotherapy. This takes several weeks and includes dynamic ortheses. The operated hand needs an extension splint, even at nighttime. According to the original description of the method, contractures distal to the metacarpophalangeal joints always require a second procedure. Nevertheless, full thickness skin grafts may have to be used to cover transverse defects at the digital flexion creases. The applicability of this method is limited because it requires a particularly cooperative and intelligent patient.

The disadvantages of dermatofasciectomy are possible graft loss and the inevitable immobilization of the extremity on a plaster splint for about 10 days. Initially, full thickness skin grafts are insensate. Sometimes, long-term sensibility training (Dellon, 1988, 1995) is necessary to improve the sensory regain and static, respectively dynamic two-point discrimination in the grafted areas.

Eventually, the moving two-point discrimination in grafted areas will be twice the value of healthy control fingers. Only the pulpa sensation of healthy and operated finger shows similar dynamic two-point discrimination of 5 mm to 6 mm (Hall et al., 1997).

Although dermatofasciectomy has a low recurrence rate, the procedure cannot prevent the progression of Dupuytren's disease to the previously unaffected, non-operated surrounding areas.

Regrettably, there is no significant evidence that the amount of Dupuytren's tissue excised correlates with the final functional outcome in the various types of fasciectomy—excluding dermatofasciectomy (Orlando et al., 1974; Wylock, 1997).

In addition, none of the aforementioned techniques can achieve a true cure. That is why it is important not to disturb anatomical features and to alter their physiology to the smallest possible extent (McGrouther, 1999).

More extensive fascial excision does not necessarily yield a lower rate of relapse or progression (extension; [Roush et al., 2002]). The recurrence rates for partial fasciectomies are 12.5 to 66%, for total fasciectomies they are 21.4 to 39.7%. Progression occurs in 19.4%-51%. The lowest recurrence rate is found underneath full thickness grafts after dermatofasciectomy (Brenner et al., 2001; 2002).

With regard to preoperative extension techniques, Piza-Katzer et al.'s (2000) intermittent continuous pneumatic elongation, followed by conventional partial fasciectomy, seems the most appropriate method, compared with the similar "stretching" techniques (Messina, 1991, 1994; Citron et al., 1998).

For comparison: 13 patients treated by Citron et al. (1998) with four weeks of extension achieved an improvement of their range of motion from 123 degrees to 175 degrees after 18 months; but five patients later developed algodystrophy with stiff fingers, pain, and "autonomic dysregulation". In two additional patients, relapses were recorded. One patient of 8/13 cases with complications developed an extension contracture because of flexor tendon adhesions, which required two-stage tendon grafting.

In contrast, Piza-Katzer (2000) after intermittent pneumatic distraction (IPD), describes an extension regain of 110 degrees. The only complication was delayed wound healing in one patient. In the control group (traditional treatment), however, complications occurred in 5/10 patients. Residual contractures of 18 degrees in the "stretching" group, contrasted with 35 degrees in conventionally treated controls. The period of hospitalization was almost identical.

Although Dupuytren's disease is a familial systemic, fibroproliferative disorder with polytopic fibrous deposits of seemingly autosomal dominant transmission, its multifactorial nature has been described (McFarlane, 1997; Frey, 1997; Benson, 1998).

As epidemiological investigations on Dupuytren's disease show, there is a north/south gradient which can be partly explained by the populations' different patterns of food consumption: Citrus fruit, unsaturated fatty acids of native olive oil, and soy beans are said to contain specific scavengers, which explain the low incidence of fibromatosis in, for example, Mediterranean and Asian populations.

In addition to alimentary prophylaxis, vitamins A, C, D, and E are also reputed to have a protective function (Badalamente et al., 2000).

We also know from rabbit experiments that an overdose of 1,000 IU/kg (corresponding to 675 mg

alpha-tocepherol) per day produced vasodilatation mediated by epithelium derived relaxing factor, whereas capillary relaxation decreased appreciably after maximum doses of 10,000 IU/kg. Overdoses of antioxidants can disturb the expected balance as much as a lack of scavengers can, making a toxicological reassessment of alimentary and pharmacological antioxidants desirable (KEANEY et al., 1994; KOCH, 1996).

It would be nice to have an antioxidant that acts as a scavenger in Dupuytren's disease, or a drug with few side effects that inhibits connective tissue proliferation, prevents its unbridled progression, and thus makes fasciectomy unnecessary.

The initial efforts at a systemic treatment of the systemic connective tissue process, which we call Dupuytren's disease, was done with allopurinol, a scavenger, and had results (MURRELL et al., 1987). High systemic doses of steroids also seem successful in inhibiting the fibroproliferation in Dupuytren's disease.

Because of its reducing properties, the semimetal selenium possesses a detoxifying effect (EKSNER et al., 1987). Coconuts are well known for their high selenium concentrations.

The use of inhibitors of synthesis, receptor blockers, and inhibitors of cell-matrix interactions, such as integrins, is another pharmacological option (QUAGLINO et al., 1997).

We believe, that interferon (IFN gamma, and IFN-alpha 2b) may be another agent that could provide successful causative therapy in Dupuytren's disease . Interferon is a cytokine produced in T-helper lymphocytes. Interferon blocks the synthesis of growth factors (transforming growth factor subunit beta and others), blocks the expression of smooth muscle actin and the aggregation of the fibronexus complex, and thus generally minimizes the ability of myofibroblasts to contract (SANDERS et al., 1999; THOMASEK, 1999). The antiproliferative effect of intralesional injections of gamma-interferon on hypertrophic scars and Dupuytren's disease has been clinically proven (PITTET et al., 1994).

Nifedipine and verapamil inhibit the contraction of fibroblasts in vitro (RAYAN et al., 1996).

Further studies on the cytogenetic origin of connective tissue should bring improvements for the management of Dupuytren's disease.

Due to the rapid progress in biotechnology, geneticists will be able to treat and eradicate some of the familial diseases by means of genetic engineering. Thus there is hope that Dupuytren's disease, which is obviously a familial connective tissue disease, will also be treatable by somatic gene therapy very soon (MCFARLANE, 1997).

Currently, surgical removal of the involved fascial structures remains the best treatment available.

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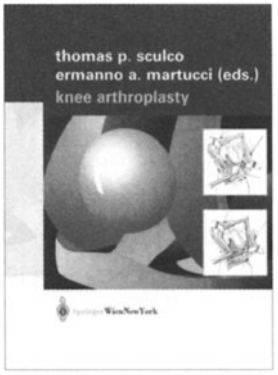

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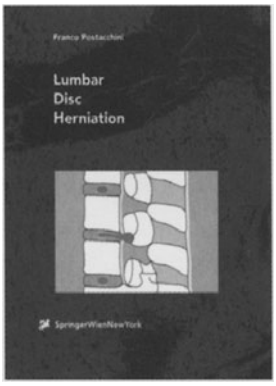

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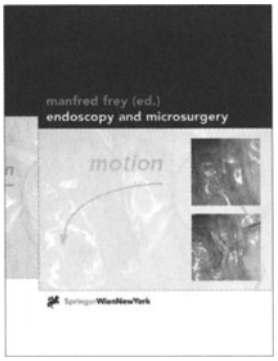

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